DISSERTATION ON

A COMPARATIVE STUDY OF EPIDURAL BUTORPHANOL AND EPIDURAL FENTANYL AS ADJUVANTS TO BUPIVACAINE IN LOWER ABDOMINAL SURGERIES

Dissertation submitted to

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfilment of the regulations for

the award of the degree of

ANAESTHESIOLOGY

M.D. BRANCH - I

THANJAVUR MEDICAL COLLEGE,

THANJAVUR - 613 004.

THE TAMILNADU DR.MGR MEDICAL UNIVERSITY

CHENNAI - 600 032.

APRIL 2015

CERTIFICATE

This is to certify that this dissertation entitled **"A COMPARATIVE STUDY OF EPIDURAL BUTORPHANOL AND EPIDURAL FENTANYL AS ADJUVANTS TO BUPIVACAINE IN LOWER ABDOMINAL SURGERIES "** is a bonafide original work of **Dr.P.S.RAJESWARI** in partial fulfilment of the requirements for M.D Branch -I (Anaesthesiology) Examination of the Tamilnadu Dr.M.G.R. Medical University to be held in APRIL - 2015. The period of study was from JUNE 2012 - JULY 2014

Prof.Dr, K.MAHADEVAN,M.S.,
DEAN
THANJAVUR MEDICAL COLLEGE
THANJAVUR- 6130004

Prof. Dr. R.MUTHUKUMARAN MD,DA
HEAD OF THE DEPARTMENT
DEPT. OF ANAESTHESIOLOGY
THANJAVUR MEDICAL COLLEGE
THANJAVUR- 613004

DECLARATION

I, **Dr.P.S.RAJESWARI**, solemnly declare that dissertation titled **"A COMPARATIVE STUDY OF EPIDURAL BUTORPHANOL AND EPIDURAL FENTANYL AS ADJUVANTS TO BUPIVACAINE IN LOWER ABDOMINAL SURGERIES"** is a bonafide work done by me at Thanjavur Medical College, Thanjavur during June 2012 to July 2014 under the guidance and supervision of **Prof. DR. R.MUTHUKUMARAN MD,DA** Department of Anaesthesiology, Thanjavur Medical College, Thanjavur.

This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University towards partial fulfilment of requirement for the award of **M.D degree (Branch -I) in Anaesthesiology**

Place: Thanjavur

Date: **(Dr.P.S.RAJESWARI)**

ACKNOWLEDGEMENT

I am extremely grateful to **Dr.K.MAHADEVAN, M.S**., **The Dean** ,Thanjavur Medical College for permitting me to do this dissertation at Thanjavur Medical College Hospital, Thanjavur.

I am indebted greatly to my Professor and Head of the Department, Department of Anaesthesiology**, Dr.R.MUTHUKUMARAN MD,DA.,** who had inspired, encouraged and guided me in every step of this study.

I express my heartiest thanks and sincere gratitude to **Dr.MOHAMED SAMEER MD,** Assistant Professor, Department of Anaesthesiology , Thanjavur Medical College for his help and suggestions for performing my study.

I sincerely thank all my Assistant Professors, Department of Anaesthesiology for their support during my study.

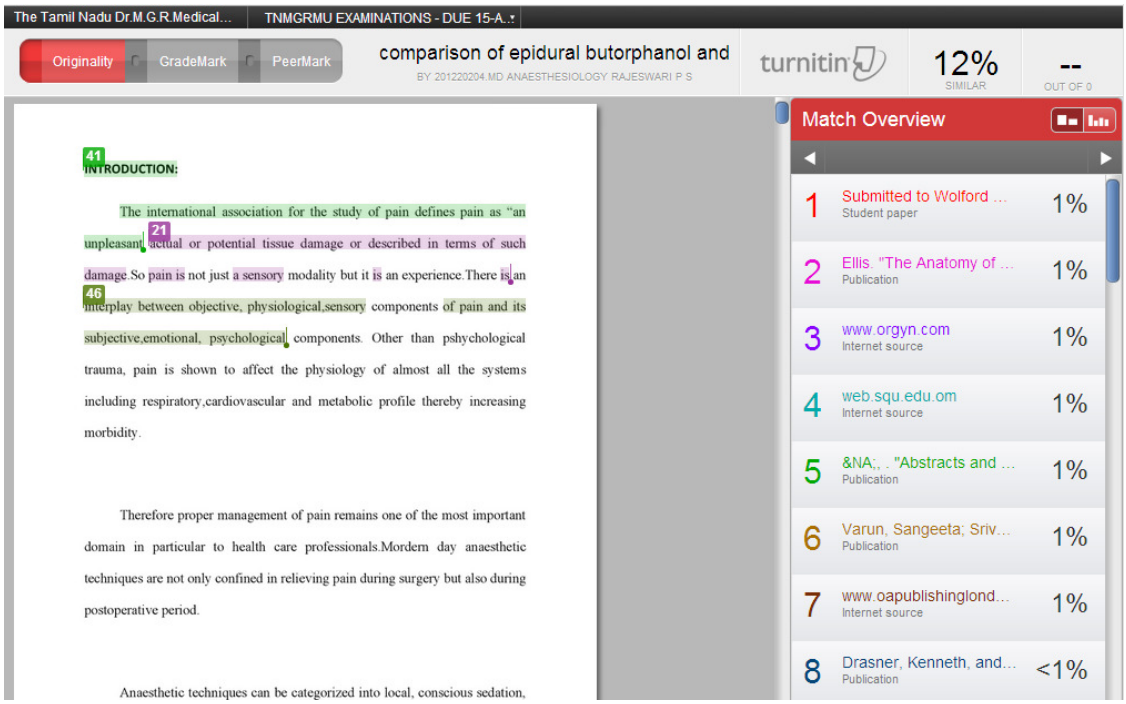
I owe my thanks to my co-post graduates for their support during the study.

I would like to acknowledge the assistance rendered by non medical assistants and the technical staffs who helped me to perform the study.

I am grateful to all my patients and volunteers who participated in this study. I owe my special thanks to my family members for their moral support in conducting the study.

Above all, I dedicate my sincere thanks and prayers to the DIVINE FORCE which guides me throughout my life towards the best.

ANTIPLAGIARISM ORIGINALITY CERTIFICATE

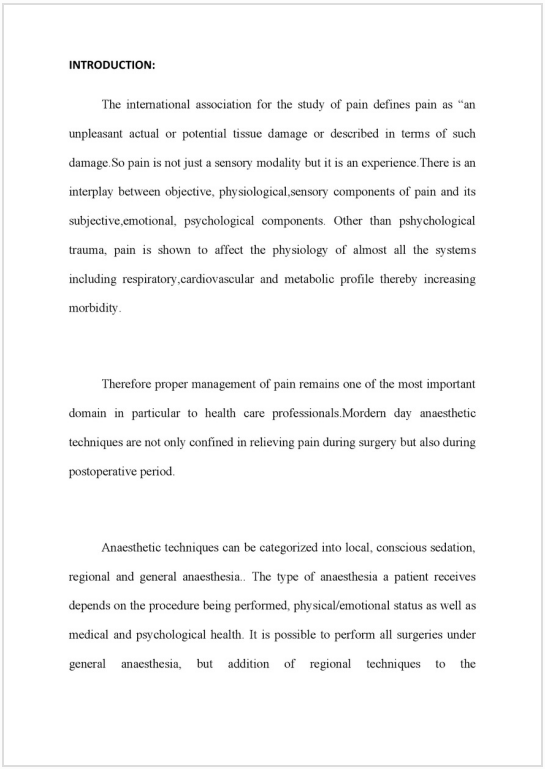


Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	201220204.md Anaesthesiology RAJ..
Assignment title:	TNMGRMU EXAMINATIONS
Submission title:	comparison of epidural butorphanol …
File name:	thesis_main_rajee_Autosaved.docx
File size:	1.38M
Page count:	102
Word count:	12,915
Character count:	74,583
Submission date:	17-Sep-2014 11:13PM
Submission ID:	453319066



INTRODUCTION:

The international association for the study of pain defines pain as "an unpleasant actual or potential tissue damage or described in terms of such damage.So pain is not just a sensory modality but it is an experience.There is an interplay between objective, physiological,sensory components of pain and its subjective,emotional, psychological components. Other than pshychological trauma, pain is shown to affect the physiology of almost all the systems including respiratory,cardiovascular and metabolic profile thereby increasing morbidity.

Therefore proper management of pain remains one of the most important domain in particular to health care professionals.Mordern day anaesthetic techniques are not only confined in relieving pain during surgery but also during postoperative period.

Anaesthetic techniques can be categorized into local, conscious sedation, regional and general anaesthesia.. The type of anaesthesia a patient receives depends on the procedure being performed, physical/emotional status as well as medical and psychological health. It is possible to perform all surgeries under general anaesthesia, but addition of regional techniques to the

CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

1. PR- Pulse Rate
2. HR- Heart Rate
3. RR- Respiratory Rate
4. SPO_2- Oxygen Saturation
5. RS- Respiratory System
6. CVS- CardioVascular system
7. CNS- Central nervous System
8. SAB- Subarachnoid Block
9. RA- Regional Anaesthesia
10. EA- Epidural Anaesthesia
11. BB- Bupivacaine- Butorphanol
12. BF- Bupivacaine- Fentanyl
13. VAS- Visual Analogue Scale
14. PCA- Patient Controlled Analgesia
15. NSAIDs- Non-Steroidal Anti-Inflammatory Drugs
16. mins- minutes
17. hrs- hours
18. mmHg- millimetres of mercury
19. mg- milligrams
20. µg- micrograms
21. LOR- Loss of resistance
22. NS- Non Significant
23. S- Significant

ABSTRACT

A COMPARATIVE STUDY OF EPIDURAL BUTORPHANOL AND EPIDURAL FENTANYL AS ADJUVANTS TO BUPIVACAINE IN LOWER ABDOMINAL SURGERIES:

Background:

Epidural opioids acting through the spinal cord receptors improve the quality and duration of analgesia along with dose-sparing effect with the local anesthetics. The present study compared the efficacy and safety profile of epidurally administered butorphanol and fentanyl combined with bupivacaine (B).

Materials and Methods:

A total of 60 adult patients of either sex of American Society of Anesthesiologist physical status I and II, aged 20-60 years, undergoing lower abdominal surgeries under epidural anesthesia were enrolled into the study. Patients were randomly divided into two groups of 30 each: bupivacaine + butorphanol (BB) and bupivacaine + fentanyl (BF). Total volume of 20ml of 0.5% bupivacaine was administered epidurally in both the groups with the addition of 1 mg butorphanol in BB group and 100 μg fentanyl in the BF group. The hemodynamic parameters as well as various block characteristics including onset, completion, level and duration of sensory analgesia,quality of analgesia as well as onset and completion of motor block were observed and compared. Adverse events and post-operative

visual analgesia scale scores were also noted and compared. Data was analyzed using Chi-square test and Fisher's exact test. Value of $P < 0.05$ was considered significant and $P < 0.001$ as highly significant.

Results:

The demographic profile of patients was comparable in both the groups. Onset and completion of sensory analgesia was earliest in BB group, followed by BF group but was not statistically significant. The duration of analgesia was significantly prolonged in BB group followed by BF which was statistically significant.

Addition of butorphanol and fentanyl to Bupivacaine had no effect on the time of onset and completion of motor block. No serious cardio-respiratory side effects were observed in any group.

Conclusions:

Butorphanol and fentanyl as epidural adjuvants are equally safe and provide comparable stable hemodynamics, early onset and establishment of sensory anesthesia. Butorphanol provides a significantly prolonged post-operative analgesia.

Key words: Bupivacaine, butorphanol, epidural anesthesia, fentanyl, lower abdominal surgeries.

INTRODUCTION

The international association for the study of pain defines pain as "an unpleasant actual or potential tissue damage or described in terms of such damage.So pain is not just a sensory modality but it is an experience.There is an interplay between objective, physiological,sensory components of pain and its subjective,emotional, psychological components. Other than pshychological trauma, pain is shown to affect the physiology of almost all the systems including respiratory, cardiovascular and metabolic profile thereby increasing morbidity[1].

Therefore proper management of pain remains one of the most important domain in particular to health care professionals.Modern day anaesthetic techniques are not only confined in relieving pain during surgery but also during postoperative period.

Anaesthetic techniques can be categorized into local, conscious sedation, regional and general anaesthesia.. The type of anaesthesia a patient receives depends on the procedure being performed, physical/emotional status as well as medical and psychological health. It is possible to perform all surgeries under general anaesthesia, but addition of

regional techniques to the anaesthesiologist's armamentarium adds flexibility and skills that benefits the patients intra and post-operatively.

Epidural anaesthesia/analgesia is one of the best accepted and most commonly employed technique in modern anaesthesiology for lower abdominal, pelvic, perineal, thoracic and lower limb surgeries.It provides surgical anaesthesia as well as postoperative analgesia.Any peripheral tissue injury including surgery leads to long term alterations in pain pathway leading to reduction in pain threshold and amplification of pain response[2].

Postoperative pain treatment should be an integral part of routine surgical and anaesthetic management both for humanitarian reasons and to reduce morbidity, associated complications as well as to accelerate rehabilitation. Good perioperative analgesia is an important avenue to attenuate surgical stress response[3].

Epidural anaesthesia provides good operative conditions with good sensory and motor blockade, contracted bowels retaining adequate spontaneous respiration, hemodynamic stability and facilities for postoperative analgesia. Discovery of opioid receptors in the spinal cord and subsequent development of epidural/intrathecal administration of

opioid has opened a new horizon in pain management in the perioperative period.

Bupivacaine is widely used in epidural anaesthesia. It is an amide local anaesthetic with asymmetric carbon atom.This drug is widely used for epidural anaesthesia and analgesia because of its long duration of action and differential blockade in lower concentrations.

Fentanyl is an phenyl piperidine derivative and synthetic opioid agonist with rapid onset and short duration of action. It is 75 – 125 times more potent than morphine.

Butorphanol tartarate is a synthetic opioid agonist and anagonist with analgesic potency 4-8 times that of morphine. It is considered safe than pure opioid agonist because of its ceiling effect on respiratory depression,lower addiction potential with sedation comparable to or more than morphine, lesser incidence of nausea,vomiting,pruritis which is desirable in the post operative period[3].

The present study was designed to compare epidural bupivacaine with butorphanol and epidural bupivacaine with fentanyl in lower abdominal surgeries.

AIMS AND OBJECTIVES OF THE STUDY

This study aims to compare the efficacy of butorphanol and fentanyl added as adjuvants to bupivacaine in epidural anaesthesia for elective lower abdominal surgeries. The following points would be considered for comparison:

1.Onset and completion of sensory blockade

2.Level of sensory block

3.Quality of analgesia

4.Duration of analgesia

5.Sedation scores

6.Side effects

EPIDURAL ANAESTHESIA

HISTORY OF EPIDURAL ANAESTHESIA[4,5]:

Jean Enthuse Sicard and Ferdinand in 1901 were the first to approach epidural space by caudal route with cocaine. Fidel Pages, a Spanish military surgeon in 1921 described his work with lumbar epidural anaesthesia and is considered as the father of modern epidural anaesthesia. In 1931, Archile Dogliotti, an Italian surgeon popularized segmental epidural anaesthesia and described the loss of resistance technique. In 1949, Martinez Curbelo, an Cuban anaesthesiologist practiced continous epidural anaesthesia using Tuohy-Huber needle and silk ureteral catheter.

ANATOMY OF EPIDURAL SPACE[6]

A good understanding of spinal column, spinal canal and spinal ligaments through which the epidural needle passes is required for the success of epidural anaesthesia.It is by practice and experience that the anaesthesiologist will able to appreciate the resistance offered by the ligaments as the epidural needle advances through the epidural space.

Epidural space can be defined as the area outside the dural sac but inside the spinal canal, extending from the foramen magnum to the sacrococcygeal ligament.

Epidural space is discontinous and is divided into posterior, lateral and anterior compartments. The posterior epidural space is of greatest relevance to the anaesthesiologist because this is where the epidural needle is placed in epidural anaesthesia/analgesia.

BOUNDARIES OF THE EPIDURAL SPACE:

The epidural space is bounded

1.Cranially by the foramen of magnum

2.Caudally by the saccrococcygeal ligament covering the sacral hiatus

3.Anteriorly by the posterior longitudinal ligament

4.Laterally by the vertebral pedicle

5.Posteriorly by the ligamentum flavum and vertebral lamina

Epidural space is most shallow anteriorly and deepest posteriorly.The antero-posterior dimension of the posterior epidural space is largest at mid lumbar levels,decreases at thoracic levels and disappears above C7.

CONTENTS OF THE EPIDURAL SPACE:

The contents of the epidural space are:

1.Loose areolar tissue

2.Epidural fat: Most widespread material is fat predominantly located in the lateral and posterior epidural space with clinical importance in pharmacokinetics of epidurally administered drugs.

3.31 pairs of spinal nerves with their dural cuff on their way to the intervertebral foramen.

4.Sacral and coccygeal nerves

5. Spinal arteries arising from different sources at different levels enter the epidural space through the intervertebral foramen and supply the spinal cord,meninges, periosteum and ligaments.

6. Vertebral venous plexus: A network of veins, the Batson venous plexus courses through the anterior and lateral parts of epidural space.These venous plexus communicates above with the intracranial venous sinuses, below with pelvic, portal and caval systems and also with intervertebral veins.

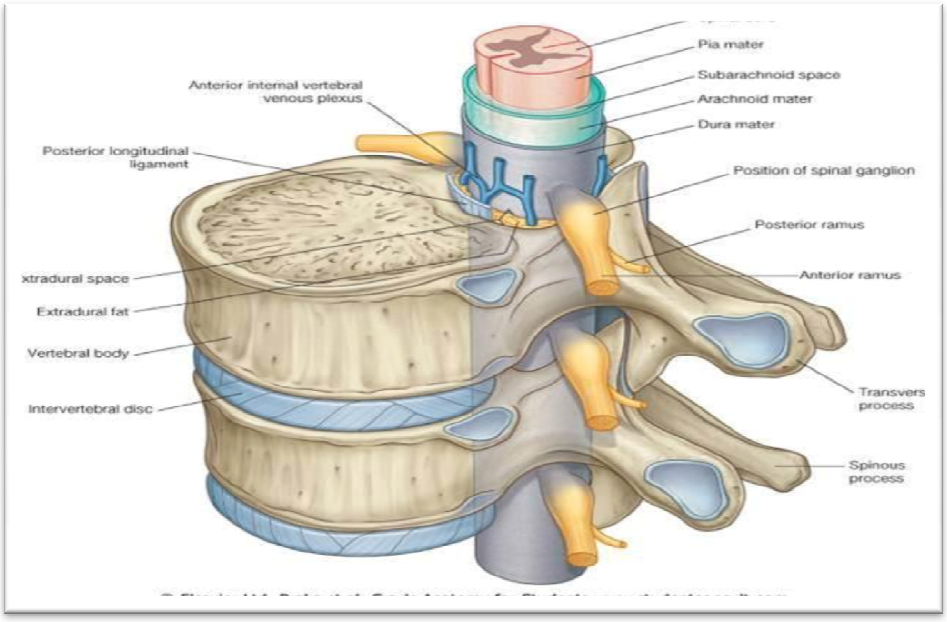


FIG 1: EPIDURAL SPACE- BOUNDARIES

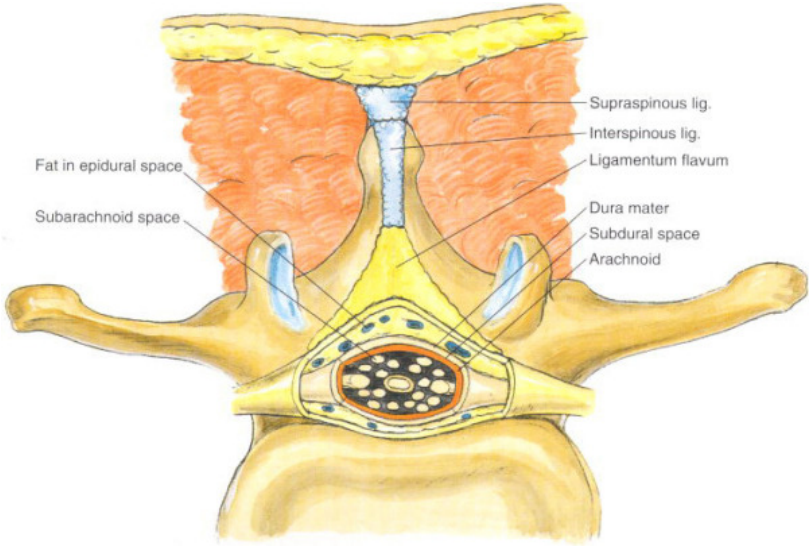


FIG 2A: EPIDURAL SPACE- CROSS SECTIONAL ANATOMY

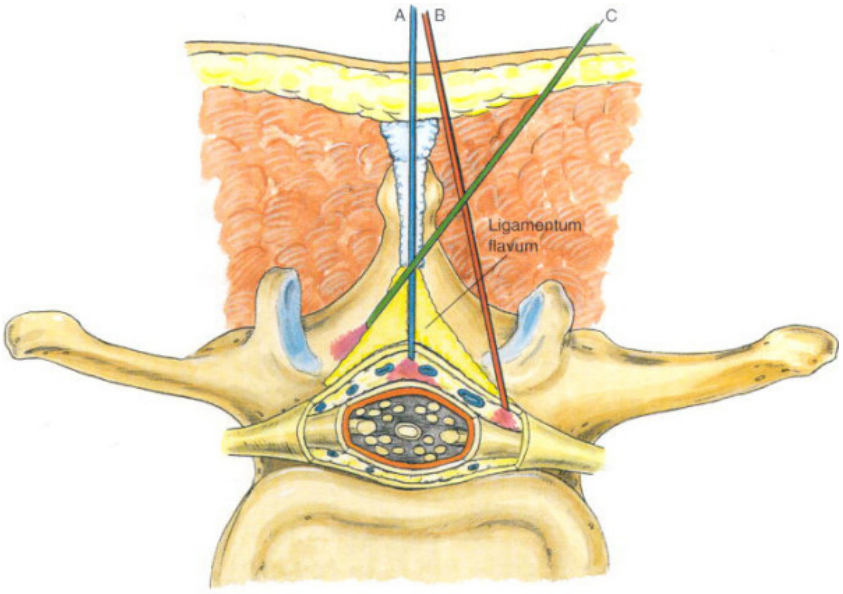


FIG 2B: EPIDURAL SPACE- CS SHOWING THE ORIENTATION OF EPIDURAL NEEDLE IN VARIOUS PLANES

Ligamentum flavum is an important landmark while identifying epidural space using epidural needle because penetrating this tissue produces a noticeable loss of resistance to air or liquid. This point of resistance should be identified because any further advancement results in inadvertant dural puncture. The ligamentum flavum will be perceived as a thicker ligament if the needle is kept in the midline . It is to maintain the midline position of the epidural needle (*needle A)* during lumbar epidural techniques. If an oblique approach is taken, a "false release" can be produced *(needle C)* or the perception of a thin ligament can be reinforced *(needle B)*[7].

EPIDURAL SPACE AND NEGATIVE PRESSURE:

In 1828 Heldt and Maloney described about negative extra dural pressure.Several factors contribute to the generation of subatmospheric pressure in the epidural space.The natural effect of Starling forces across the capillary walls produce a low fluid pressure in the tissues on the basis of oncotic pressure.This results in subatmospheric pressure and tissue collapse in the spaces of opposing surfaces of spinal canal, including the planes where dura opposes the epidural fat or canal wall, and between epidural fat and canal wall.

The pressure in the epidural space is important for several reasons. First, the hanging drop method used to identify entry into the epidural space directly relies on recognition of subatmospheric pressure in the spinal canal.Secondly recent reports indicate that presence of epidural wave forms will reliably confirm needle placement in the epidural space.Lastly increase in epidural pressure above its natural subatmospheric pressure by infusion of solutions may cause adverse effects by displacing the CSF and raising ICT.

MECHANISM OF ACTION OF LOCAL ANAESTHETICS IN EPIDURAL SPACE[7]:

The principle site of action of local anaesthetics is that portion of nerve roots in the epidural space as the nerves emerge from the dura and then pass into intervertebral foramen. Also substantial amount of drug diffuse into the subarachanoid space for its action. But the final destination of epidurally administered drug is that they must be absorbed into the blood stream, which occurs rapidly from the epidural space.

The proposed site of action of local anaesthetics in the epidural sapce:

1. The ink cuff zone- spinal nerve roots with their dural sleeves
2. Mixed spinal nerves at the intervertebral foramen
3. Dorsal root ganglia
4. Substance of the spinal cord

The major routes of elimination of local anaethetics from epidural space is uptake by:

1.Blood vessels

2.Extra dural fat

3.Nerve tissue

PHYSIOLOGICAL EFFECTS OF EPIDURAL BLOCKADE [2,7,8]:

1.CARDIOVASCULAR EFFECTS:

Epidural anaesthesia/analgesia has primarily indirect effects on cardiovascular system.Drugs absorbed into systemic circulation from the epidural space may have direct effects on organ systems. The indirect effects are mediated primarily through blockade of sympathetic nervous system and include reflex response to the primary cardiovascular effect. The extent of effect of neuraxial blockade on CVS depends on the number of spinal segments blocked. Most notable effect is systemic hypotension. Blockade below T4 results in dilatation of arteries and venous capacitance vessels, leading to decreased SVR and decreased venous return. The barorecptors are activated to produce vasoconstriction of the upper extremities. Blockade above T4 interrupts cardiac sympathetic fibres leading to bradycardia, decreased cardiac output and profound systemic hypotension.

2.PULMONARY EFFECTS:

Epidural anaesthesia /analgesia has minor effects on respiratory physiology and these changes have minimal consequences in other wise healthy persons. During high thoracic epidural anaesthesia vital capacity decreases by 6%, total lung capacity decreases by 3.5% and the absolute

value of FEV1 decreases by 5%, FEV1/FVC ratio and tidal volume remain unchanged.Dense epidural anaesthesia does not affect at-rest respiratory function, but the ability to cough and clear secretions may be adversely affected.

Gas exchange and ventilation-perfusion matching remained unchanged in elderly patients after lumbar epidural anaesthesia.Neither lumbar nor thoracic epidural anaesthesia impair the ventilatory response to hypercarbia or hypoxia in elderly patients.

3.NEUROENDOCRINE EFFECTS:

Neuraxial blockade may influence nuroendocrine functions. Neuraxial blockade higher than T9-10 is associated with decreased plasma epinephrine and norepinephrine levels.Neuraxial blockade may contribute to a mitigated stress response by direct blockade of both afferent and efferent signals. Epidural anaesthesia results in an increased tissue oxygenation compared to GA.

Neuraxial blockade ,although attenuated, does not inhibit the increases in intra operative stress hormones like glucose,cortisol, catecholamines and ACTH.

4.VISCERAL EFFECTS:

a. Bladder: Bladder is innervated by nerves travelling with S2 through S4 nerve roots. Sympathetic nerves to bladder originate at the low thoracic /high lumbar levels and parasympathetic innervation originates at the sacral levels. Blockade of these nerve roots directly affect detrusor muscle and urinary sphincter function resulting in urinary retenion.

b.Intestines: The sympathetic supply to abdominal viscera originates from T6 to T12-L1, while the parasympathetic supply to the gut is via the vagus nerve.

Therefore,neuraxial blockade at the mid- low thoracic levels result in sympathetic denervation and parasympathetic dominance, resulting in contracted gut,relaxed sphincters and normal peristalsis.

5.THERMOREGULATION:

Thermoregulation is impaired in neuraxial blockade,the predominant cause being decreased core temperature resulting from redistributiion of heat from core to periphery due to peripheral vasodilation.In addition, central thermo-regulatory response is also impaired. Sympathetic amd motor blockade preclude vasoconstriction and shivering in the lower part of the body, but even in the upper half of the body, neuraxial blockade lower thermoregulatory vasoconstriction and shivering thresholds.

6.CENTRAL NERVOUS SYSTEM:

Cerebral blood flow is auto regulated;thus blood flow to central nervous system remains constant during neuraxial anaesthesia unless there is profound hypotension i.e., MAP <55mmHg in a normotensive individual.Sedation scores were directly related to the extent of segmental blockade .Sedation in neuraxial anaesthesia is due to decreased afferent input to the RAS.Neuraxial anaesthesia may be associated with a decreased incidence of early postoperative cognitive dysfunction.

7.DIFFERENTIAL BLOCKADE:

The ability to block sensory,motor and sympathetic nerve functions to varying degree is called 'differential blockade'. Although smaller and myelinated fibres are generally said to be blocked more easily than larger and unmyelinated ones, the phenomenon of differential blockade appears to be more complex, especially for neuraxial anaesthesia. Sympathetic impulses carried by smaller C fibres are blocked earlier than larger sensory and motor fibres.As a result, level of sympathetic blockade is 2 to 6 segments above the level of sensory blockade,which inturn is two segments higher than motor blockade.

INDICATIONS FOR EPIDURAL ANAESTHESIA:

Epidural anaesthesia can be used for a variety of surgeries extending from neck to foot.

COMMON APPLICATIONS OF EPIDURAL ANAESTHESIA:

The common applications of epidural anaesthesia are:

1.Prolonged orthopedic surgeries like major hip/knee surgeries, repair of pelvis fractures

2.Obstetrics and gynaecological surgeries like C-section, labour analgesia

3.Urological surgeries involving prostrate, bladder and ureters

4.Epidural analgesia for upper abdominal and thoracic surgeries

5.Epidural anaesthesia for lower abdominal and lower limb surgeries

6.In high risk patients with respiratory problems

7.Paediatric caudal for lower abdominal and lower limb surgeries.

CONTRAINDICATIONS FOR EPIDURAL ANAESTHESIA:

ABSOLUTE:

1. Patient refusal
2. Infection at the site of planned needle puncture
3. Elevated intracranial tension
4. Bleeding diasthesis

RELATIVE:

1. Sepsis
2. Uncooperative patient
3. Preexisting neurological deficits
4. Stenotic valvular lesions
5. Severe spinal deformity

PREPARATION FOR NEURAXIAL ANAESTHESIA [9,10]:

1.EMERGENCY DRUGS AND SUPPLIES: Adverse events are fortunately relatively infrequent,however immediate action must be taken when they occur in order to mitigate or prevent severe complications. The area where the block is to be performed must be equipped with an oxygen source,a means to administer postive pressure ventilation, equipment for airway management as well as emergency drugs.

2.PATIENT PREPARATION: Patient preparation procedures that apply to general anaesthesia apply to neuraxial anaesthesia as well. A thorough preoperative evaluation should be performed, patient's starvation status should be greater than 6hours for solids, and greater than 2hours for clear liquids.Informed written consent should be obtained and finally a back up plan in the event of failed neuraxial anaesthesia should be discussed.

3.MONITORING: During the performance of neuraxial anaesthesia monitoring should include recording the baseline level of consciousness, continuous pulse oximetry and heart rate monitoring, non-invasive blood pressure monitoring and continuous ECG monitoring. Intraoperative monitoring should include ASA's standard monitoring including temperature monitoring.

4.SEDATION: Light sedation may be administered before initiation of neuraxial anaesthesia, if this is desired by the patient and deemed appropriate by the anaesthesiologist. If spinal or epidural block is performed in the preoperative holding area and neuraxial drugs have been injected or sedation has been administered the patient should not be left alone or unmonitored.

5.PATIENT POSITIONING: Careful attention to patient positioning is critical to successful epidural blockade.Epidural anaesthesia can be performed in the sitting,lateral decubitus ,or jack knife position.The most comfortable position is likely to be the position in which the anaesthesiologist can perform the procedure in the smoothest and fastest way.

Most commonly used position is lateral decubitus position because it is less dependant on a well trained assistant and allows administration of sedation during procedure.Patients back should be positioned at and parallel to the edge of the bed or operating table so that the patient is within easy reach of the anaesthesiologist. The knees should be drawn towards the chest, and the neck should be in neutral or flexed position.The

patient should be asked to actively curve the lower back in order to flex the lumbar spine.

The sitting position allows for easier recognition of midline,especially in obese patients or those with scoliosis.When placing patients in this position a stool can be provide as a foot rest and a pillow placed in the lap.The assistant then maintains the patient in a vertical plane while flexing the patient's neck and placing the patient's arms over the pillow to open up the lumbar space.

FIG 3a: LATERAL DECUBITUS POSITION

FIG 3b: SITTING POSITION

6.SUPPLIES AND EQUIPMENTS: The ability to efficiently and safely initiate epidural anaesthesia depends on having the right equipment immediately available.Commercial epidural trays containing disposable equipements are available.Epidural anaesthesia usually involves placing an epidural catheter in the epidural space, allowing for continous anaesthesia or analgesia.

EPIDURAL NEEDLE:

Epidural needles used in conjunction with epidural catheters are usually 17 or 18 gauge, allowing 19 or 20 gauge catheter to be threaded through them. Standard epidural needles are 9cm long.Epidural needles

have different tip designs. Tuohy and Husted designs have a curved tip with a lateral facing orifice.The Crawford needle has a forward facing orifice, which facilitates threading of the epidural cathetar if the needle is expected to enter the space at a more acute angle.

FIG 4a: TUOHY'S EPIDURAL NEEDLE

FIG 4b: CRAWFORD EPIDURAL NEEDLE

EPIDURAL CATHETER:

Epidural catheters are made of plastic.There are two types of catheters: single and multi orifice.Single orifice catheters have one orifice in a single compartment at the tip, whereas the multiorifice catheters have a closed,bullet tip and several(usually 3) openings 0.5-1.5 cm from the tip.The ability to aspirate blood should the catheter tip be located intravascular is much easier in multiorifice catheters.The wire embedded arrow epidural cathetar is a single orifice catheter associated with less incidence of parasthesias and intravascular complications. Modern epidural catheters have centimetre markings along the catheter, usually between 5 and 20cm from the tip.

SYRINGES FOR LOSS-OF-RESISTANCE:

Syringes used for the loss of resistance technique to identify the epidural space were traditionally made of glass with a Leur-lock connector. Currently syringes made of plastic are used.The type of loss of resistance (LOR) syringe is largely a matter of preference.

TECHNIQUE FOR EPIDURAL ANAESTHESIA [9,10,11]:

Two approaches are usually followed, they are midline or paramedian approach. Both the approaches are performed with patient in lateral decubitus or sitting posture. Pateint position during and after injection of local anaesthetic drug plays less of a role in determining the final extent of sensory blockade, as gravity has little effect on the distribution of epidural anaesthetic solutions.

MIDLINE APPROACH:

It is the commonly used approach with greater accuracy and better success in normal patient. The patient is placed in lateral decubitus or sitting posture. The level of needle insertion is decided based on the segments to be blocked. After proper positioning, sterile skin preparation and drapping, a local anaesthetic skin wheal is raised at the point of needle insertion.The epidural needle is inserted in the midline with the dorsum of the anaesthesiologist's non injecting hand resting on the back of the patient, with the thumb and index finger holding the epidural needle hub (Bromage grip).The epidural needle with stylet is passed through the skin wheal and subcutaneous tissue to the depth of supraspinous ligament. This

allows the needle to be advanced perpendicular to the long axis of the spine in the saggital plane

The Needle is advanced through the supra spinous and inter spinous ligament(about 3cm depth) giving a gritty feel.At that point the needle sits firmly in the midline in the saggital plane with the stylet removed.If the needle shaft falls,the tip is still in the subcutaneous plane and not in the ligament. A lateral deviation implies the needle being deflected off by a spinous process. Various techniques have been described to identify epidural space but LOR to air or normal saline is commonly used, by using an LOR syringe. By applying constant pressure to the LOR syringe attached to the hub of epidural needle, loss of resistance can be appreciated once the needle tip pierces the ligamentum flavum and enters the epidural space.

PARAMEDIAN APPROACH:

This approach is indicated for patients whose spine cannot be flexed and positioned easily, patients with calcified ligaments, spinal deformities like kyphoscoliosis and while performing thoracic epidural. In paramedian approach, a skin wheal is raised 1cm lateral and 1cm caudad to the caudal edge of the cephalad spinous process.

The epidural needle is inserted 10 to 15 degrees off the saggital plane in a cephalo- medial plane. If the needle contacts the bone, it is redirected slightly ia cephalad direction. If bone is again contacted, but at a deeper level, the slight cephalad angulation is continued and the needle is walked off the lamina into the ligamentum flavum. Paraspinous muscles are pierced by the epidural needle with little resistance before piercing the ligamentum flavum and entering the epidural space.

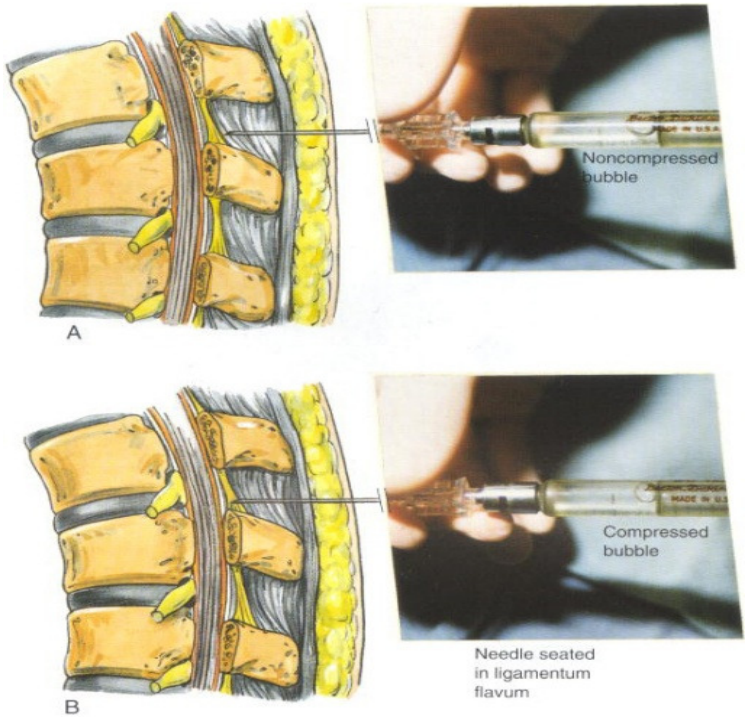


FIG 5a: LOSS OF RESISTANCE TO NORMAL SALINE

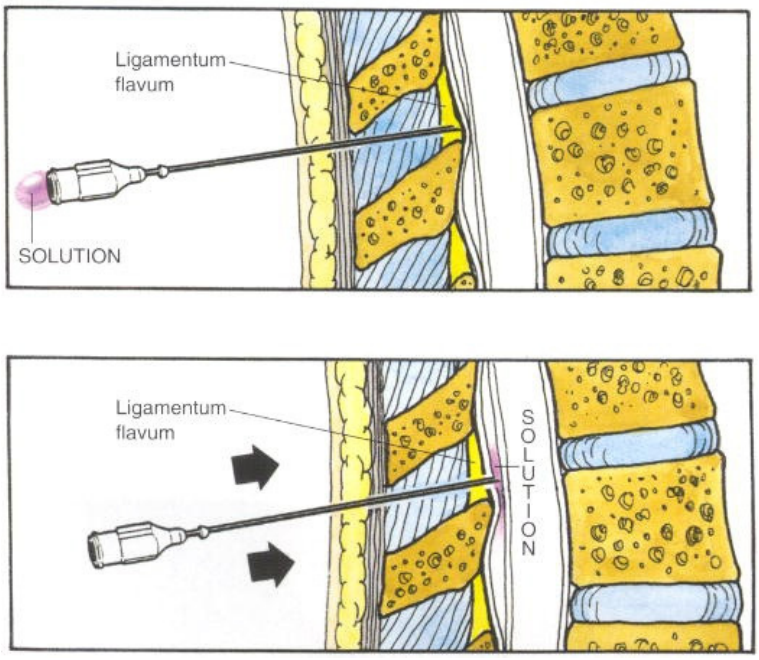


FIG 5b: NEGATIVE PRESSURE SIGN(HANGING DROP)

IDENTIFICATION OF EPIDURAL SPACE[10,11]:

Two classical methods have been described to identify the epidural space.

a) Loss of resistance to air or normal saline-The Sicard,Dogliotti test:

This technique is based on the fact that there is loss of resistance when needle enters the epidural space piercing the interspinous ligament and the ligamentum flavum. Either air or normal saline can be used to identify LOR. Recent evidences suggest several reasons for the superiority of saline compared to air.These include lower incidence of patchy epidural

blockade, pneumocephalus and PPDH if there is inadvertent dural puncture. Moreover using LOR to saline makes the change in resistance to be more accurately transmitted to the anaesthesiologist's finger as it is incompressible. But the problem is difficulty in differentiating CSF from saline if the dura is punctured.Reported complications of LOR to air includes air embolism, subcutaneous emphysema and nerve root compression.

Various mechanical aids have been incorporated to facilitate the appreciation of LOR namely MACINTOSH'S needle with spring loaded stylet, BRUNNER and ILKE'S spring loaded syringe and ZELENKA'S "U" tube with ballon indicator.

b) NEGATIVE PRESSURE SIGN:

Negative pressure in the epidural space can be appreciated by the **Hanging drop sign of Gutterrez.** By placing a drop of liquid in the hub of the needle, slowly the epidural needle is advanced into the epidural space. Liquid is placed once the needle is placed in the interspinous ligament. As the needle advances into the epidural space the hanging drop of liquid will be sucked in due to the negative pressure in the epidural space.

Various mechanical aids have been used to identify this negative pressure, namely ANEROIOD manometer, "U" tube manometer, ODOM'S indicator,BROOK'S indicator, ZORRAQUIN'S bulb indicator and DAWKIN'S gravity indicator.

EPIDURAL TEST DOSE [9]:

The traditional epidural test dose tests whether the epidural catheter is malpositioned in the blood vessel or the intrathecal space. A negative response to an epidural test dose does not guarantee the correct placement of the epidural catheter in epidural space, but rather decreases the likelihood that catheter tip is in a blood vessel or the intrathecal space. The intravascular and intrathecal test doses may either be combined in a single injection or divided. Intrathecal placement of the catheter is tested by injecting small subanaesthetic bolus dose of local anaesthetic drug, low enough so that there is minimal risk of total spinal. Intravascular placement of a catheter is traditionally tested by injecting adrenaline or subtoxic doses of local anaesthetics.

TABLE 1:

COMBINED INTRAVASCULAR & INTRATHECAL TEST DOSE		
TEST DOSE COMPONENTS	**POSITIVE INTRA - VASCULAR TEST DOSE**	**POSITIVE INTRA - THECAL TEST DOSE**
Lignocaine 1.5% with adrenaline 1:200,000, 3ml	Increase in HR by 20 beats per minute	Motor blockade by 3-5 minutes
Bupivacaine 0.25% with adrenaline 1:200,000, 3ml	Increase in HR by 20 beats per minute	Motor blockade by 3-5 minutes

TABLE 2a:INTRAVENOUS TEST DOSE:

TEST DOSE	POSITIVE INTRVASCULAR EFFECTS
LIGNOCAINE 100mg BUPIVACINE 25mg CHLORPROCAINE 90mg	TINNITUS CIRCUMORAL NUMBNESS, DIZZINESS
FENTANYL 100mcg	DIZZINESS, DROWSINESS
AIR 1 ml	MILL WHEEL MURMUR OVER RIGHT HEART

TABLE 2b:INTRATHECAL TEST DOSES:

TEST DOSE	POSITIVE INTRATHECAL EFFECT
LIGNOCAINE 40-60 mg BUPIVACAINE 7.5 mg	MOTOR BLOCKADE AT 3-5 MINUTES

COMPLICATIONS RELATED TO EPIDURAL ANAESTHESIA:

MAJOR COMPLICATIONS:

The major complications include direct trauma to nerves, systemic toxicity with inadvertent intravascular injection, subdural injection of drugs, total spinal with inadvertent dural puncture, epidural abscess, meningitis, epidural hematoma and allergy to local anaesthetics.

MINOR COMPLICATIONS:

The minor complications include backache, nausea vomiting, hearing loss, Horner Syndrome, pneumocepahlus, shivering, urinary retention and post dural puncture headache(PDPH).

PHYSIOLOGY OF PAIN [12,13,14]

Pain is protective in nature. It immobilizes the injured part and prevents further damage. Pain pathway and the nociceptive input are not passively transmitted from the periphery to the brain. The nociceptive pathway is an afferent three-neuronal ascending system, with decending modulation from cortex, thalamus and medulla. Nociceptors are free endings located in skin and muscle, bone and connective tissues with cell bodies located in the dorsal root ganglia.

The first order neurons that make up the dual ascending system have their origins in the periphery as A delta and polymodal C fibres. A delta fibres transmit first pain which is described as sharp or stinging in nature and localized, whereas the polymodal C fibres are more diffuse in nature and associated with affective and motivational aspects of pain. First order neurons synapse on the second order neurons in the dorsal horn primarily within laminas I,II and V, where they release excitatory amino acids and neuropeptides. Second order neurons include the nociceptive specific neurons NSR and WDR neurons, located in lamina I and lamina IV,V,VI respectively.

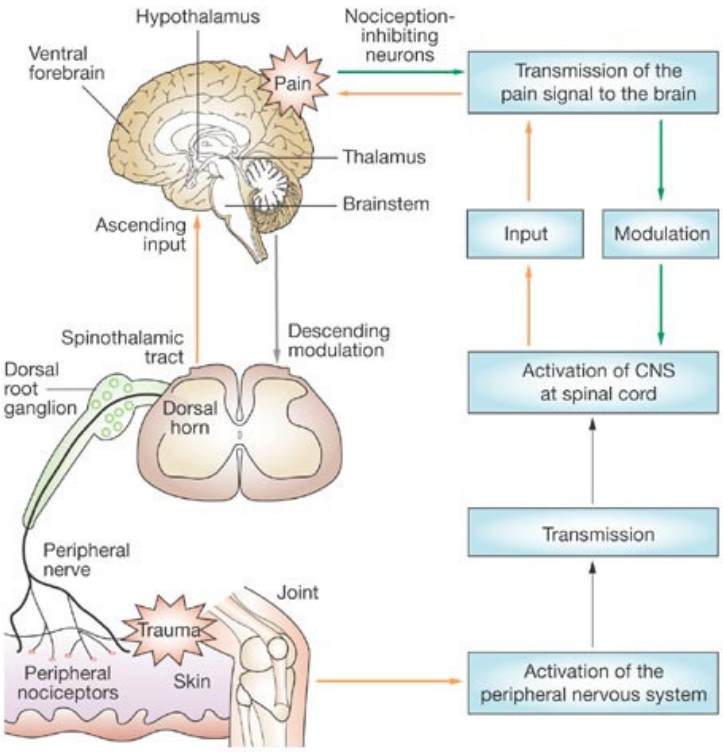


FIG 6a: THE PAIN PATHWAY

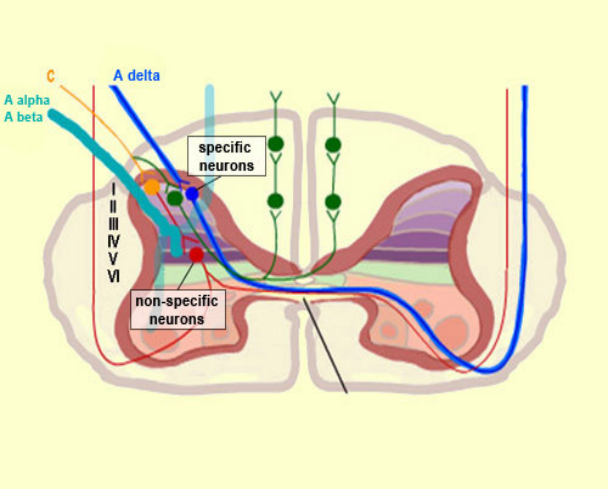


FIG 6b: THE REXED LAMINAE

PAIN PROCESSING [14,15]:

Tissue injury tends to fuel neuroplastic changes in the nervous system which results in both peripheral and central sensitization, clinically manifesting as allodynia and hyperalgesia. The four elements of pain processing include:

1.Transduction

2.Transmission

3.Modulation (inhibition/augmentation of pain signals)

4.Perception

Transduction is the process by which noxious painful stimuli are converted into actional potential. Transmission occurs when the action potential is conducted through the nervous system via the first, second and third order neurons which have their cell bodies in the dorsal root ganglion, dorsal horn and thalamus respectively. Modulation involves alteration of afferent neural transmission along the pain pathway, which may be augmentation or inhibition of pain signals. Augmentation of pain signals is manifestated as central sensitization which is a consequence of neuronal plasticity. Perception of pain is the final common pathway resulting from

integeration of painful input into the somatosensory and limbic cortices. A multi modal approach to pain therapy should target all four elements of pain processing pathway. Epidural analgesia is a critical component of multimodal preoperative pain management and improved patient outcome.

THE GATE CONTROL THEORY OF PAIN:

The observation that there was no link between stimulus and pain reported, and that pain perceived was affected by physiological and psychological variables led Melzack and Wall(1965) to propose a gate theory of pain modulation which has subsequently been modified.

Large and small fibres input into spinal cord transmission cells and into the substansia gelatinosa(lamina II of Rexed).The transmission of impulses to the transmitter cells is controlled by a spinal gating system. The is in turn influenced by the relative activity in large or small fibres. Activity in large fibres tend to inhibit transmission whereas activity in small fibres tends to stimulate transmission(opens the gate). The large diameter fibres are not necessarily nociceptive afferents; flooding the dorsal horn with cutaneous touch and pressure sensation may close the gate for smaller nociceptive input.

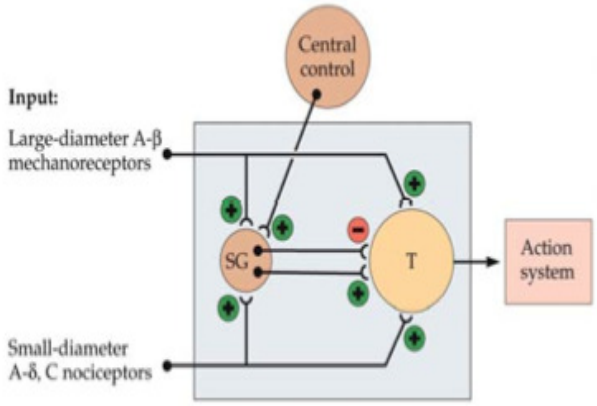


FIG 7: GATE CONTROL THEORY OF PAIN

PHYSIOLOGICAL SEQUELAE OF SURGICAL PAIN:

The neuroendocrine response to surgical pain causes increased secretion of catabolic hormones like cotrisol, glucagon, growth hormone and catecholamines, and decreased secretion of anabolic hormones. The sympathoadrenal response have detrimental effects on numerous organ systems.

1. CARDIOVASCULAR SYSTEM: Tachycardia, hypertension, increase in cardiac workload.

2. PULMONARY SYSTEM: Respiratory muscle spasm, decrease in vital capacity, atelectasis, hypoxia and increase in risk of pulmonary infection.

3. GIT: Postoperative ileus

4. RENAL: Increased risk of oliguria and urinary retention.

5.COAGULATION: Increased risk of thromboemboli

6.IMMUNE SYSTEM: Impaired immune function

7.PSYCHOLOGICAL: Anxiety, fear, frustration resulting in poor patient satisfaction.

ASSESSMENT OF PAIN [16] :

"Pain is what a patient says it is" a maxim first attributed to Dr. John Bonica, the father of pain medicine. A variety of pain meseaurement scales exist that can be helpful yet are not definitive.

UNIDIMENSIONAL SCALES: Numerical pain scale, the visual analog scale, the "faces" pain rating scale, activity tolerance scale.

MULTIDIMENSIONAL SCALES: McGill Pain Questionnaire, Brief Pain Inventory, The Breakthrough Pain Questionnaire.

1.VERBAL PAIN SCALE:

Verbal pain rating scales(none, mild,moderate,severe) have been used for many years and provide a simple tool to measure pain intensity.This scale may be used to measure treatment effect and may be more sensitive in detecting an improvement.

2.VISUAL ANALOGUE SCALE(VAS):

VAS is on of the most widely used measures of pain intensity. Its consists of 10cm line marked at one end “no pain” and at the other end as “worst pain ever” or similar phrases.The patient is asked to indicate where on the line he or she rates the pain and numerical value is given simply by measuring the distance between no pain and patient mark. Its is conveniently presented as a slide rule with one side used by the patient and other side by the assessor. Advantage of VAS is its simplicity and it is independent of language. The main disadvantage is it doesn’t differentiate affective and sensory components.

NUMERICAL PAIN RATING SCALE is similar to VAS, but with numericals 0-10, with 0 being “no pain” and 10 being “ worst pain ever” marked on a 10cm line.

3.McGILL PAIN QUESTIONNAIRE(MPQ):

Unlike VAS, the questionnaire helps to differentiate affective and sensory components of pain, providing an accurate measurement of complete pain experience.It scales pain in three dimensions- sensory, affective and evaluative. Proven to be valid and reliable tool, MPQ is widely used in both clinical and research work.

4.WEST HAVEN YALE PAIN INVENTORY:

More brief and more classical in its psychometric approach.

5.BRIEF PAIN INVENTORY:

Valid and brief multidimensional pain measurement.

6.MEMORIAL PAIN ASSESSMENT SCALE:

Scales pain, pain relief and mood on visual analog scale.

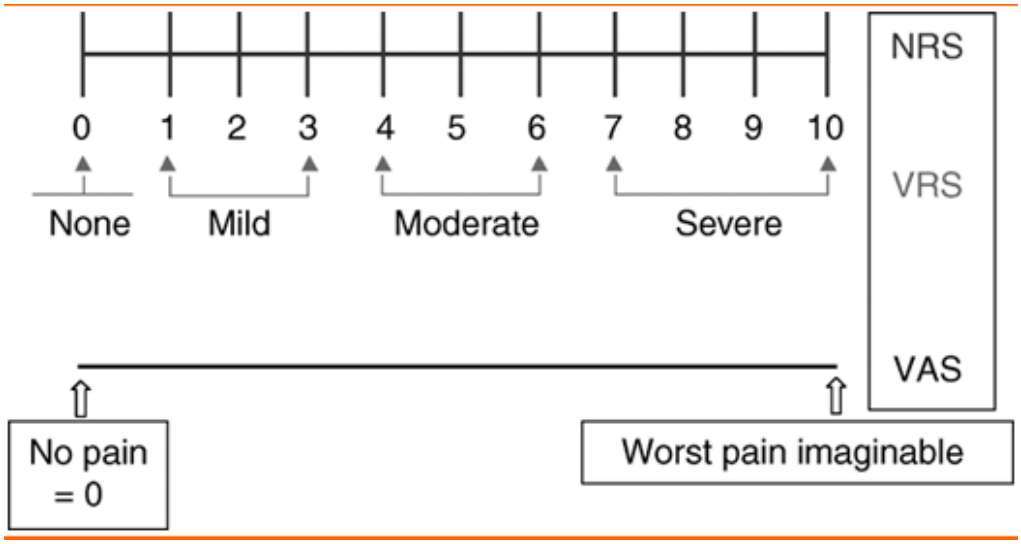


FIG 8a: NRS:Numerical pain rating scale/VRS:Verbal pain rating scale/VAS:Visual analog scale

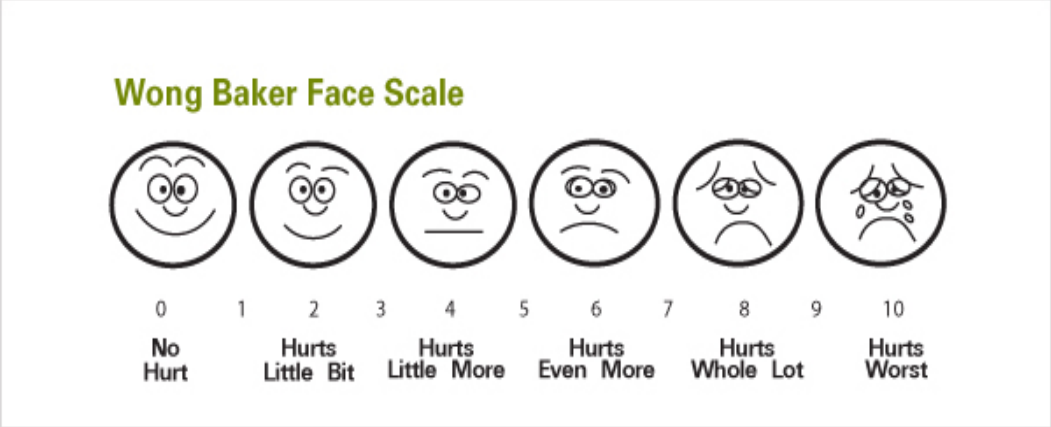


FIG 8b: WONG BAKER FACE SCALE FOR PAIN ASSESSMENT

TREATMENT MODALITIES OF PAIN MANAGEMENT [4,17]:

PREEMPTIVE ANALGESIA:

The importance of peripheral and central sensitization in pain pathway has fostered the concept of "preemptive analgesia" in patients undergoing surgery. This type of analgesia aims at effective analgesia prior to the onset of noxious stimuli such as surgical trauma. The mode of analgesia involves administration of effective opioids, NSAIDS or ketamine, central neuraxial blockade, wound infiltration with local anaesthetics. Experimental studies have shown that preemptive analgesia can attenuate peripheral and central sensitization to pain. Many studies have reported significant reduction in postoperative analgesic requirements in patients receiving preemptive analgesia athough few studies do not.

MULTIMODAL APPROACH:

It is defined as two or more analgesics agents or techniques used in combination.The ASA,"Practice guidelines for acute pain management in perioperative setting" contains the following statement.

"During the administration of anaesthetics for surgery, the needs of many patients may best be met by taking advantage of combined effect of number of agents. Similarly, there is a growing conviction that a multimodality approach in providing perioperative analgesia has

advantages over single modality approach. The literature supports the use of two or more analgesic techniques(including non pharmacological methods) used in combination for the control of perioperative pain, especially when different sites and/or mechanisms of action are involved and/or when synergy of effect is achieved. The literature also indicates that multimodality approaches are associated with side effects no greater than those resulting from single analgesic techniques."

Postoperative analgesic modalities include oral and parenteral analgesics, peripheral nerve blocks, neuraxial blocks with local anaesthetics ,opioids, adjunctive techniques like TENS and physiotherapy. Selection of analgesic technique is based on three factors: patient, procedure and the setting(inpatient or outpatient).

1.SYSTEMIC ANALGESIC TECHNIQUES:

OPIOIDS: They exert their analgesic effect through mu-receptors in the brain and some peripheral receptors. They are used for moderate to severe pain. The major advantage is that there is no analgesic ceiling effect. Analgesic is limited by the development of tolerance and their side effects like nausea,vomiting, respiratory depression and sedation. Various routes of administration are subcutaneous, oral, transcutaneous, intramuscular and intravenous. Intravenous patient controlled analgesia circumvents the

problem of interpatient, intra patient variability in analgesic needs, variability in serum drug concentrations, administrative delays contributing to inadequate post operative analgesia. IV-PCA provides good patient satisfaction and provides superior analgesia but the patients used more opioids.

NON OPIOIDS:

NSAIDS: These drugs primarily act by inhibiting the synthesis of prostaglandins by cycloxygenese. NSAIDS are used as a sole agent in mild to moderate pain and along with opioids for moderate to severe pain. Perioperative use is associated with many side effects like decreased hemostasis, delayed bone healing and osteogenesis, renal dysfunction and gastrointestinal haemorrhage,

KETAMINE: Perioperative subanaesthetic dose of ketamine reduces rescue anlagesic requirement or pain intensity. Low dose ketamine infusion does not cause hallucination or cognitive impairment, but causes side effects like dizziness, itching, nausea and vomiting which are comparable to opioids. Ketamine can be used intrathecally or epidurally, but racemic mixtures are found to be neurotoxic, hence neuraxial administration has been discouraged.

TRAMADOL:

Tramadol is a synthetic opioid with weak mu agonist activity. It is very effective in treating moderate postoperative pain and comparable in analgesic efficacy to asprin(650mg), codeine(60mg) or ibuprofen(400mg). Advantages of tramadol for postoperative analgesia imclude a relative lack of respiratory depression, major organ toxicity, depression of gastrointestinal motility and a low potential for abuse. It is contraindicated in patients on MAO inhibitors.

REGIONAL ANALGESIC TECHNIQUES [18,19]:

Regional analgesic techniques can be an epidural technique or peripheral regional analgesic technique. The analgesia provided by regional analgesia techniques are superior to systemic opioids and associated with decresed morbidity and mortality. It can be used as a single dose or continous epidural analgesia with opioid, local anaesthetics or local anaesthetic-opioid mixture. Use of local anaesthetic alone in epidural causes significant motor blockade and hypotension when compared to LA-opioid mixture. Several randomized trials show that epidural lipophilic opioids act by systemic action as there is no difference in the plasma concentrations, side effects, pain scores between intravenous fentanyl and epidural fentanyl. When combined with local anaesthetic they provide

superior analgesia, limit regression of sensory blockade and decrease the dose of local anaesthetic used.

BENEFITS OF EPIDURAL ANALGESIA:

Use of perioperative anaesthesia and analgesia especially with a local anaesthetic based analgesic solution can attenuate the pathophysiological response to surgery which may be associated with systemic opioids. A meta analysis of randomized data showed that perioperative use of neuraxial opioids reduced overall mortality by approximately 30%. Use of epidural analgesia can decrease the incidence of post operative gastrointestinal ,pulmonary and cardiac complications.

Postoperative thoracic epidural decreases the incidence of postoperative MI by attenuating the stress response, hypercoaguability and causes redistribution of coronary blood flow. A meta analysis showed that post operative thoracic epidural with local anaesthetic based solutions decreased incidence of pulmonary complication when compared to intercostals blocks, wound infiltration, inter pleural analgesia and epidural opioids. Some studies haves shown earlier return of gastrointestinal motility in patients undergoing abdominal surgeries with local anaesthetic

based solution for epidural analgesia. Epidural analgesia provides greater patient satisfaction than systemic opioids.

PERIPHERAL REGIONAL ANALGESIA:

This can be used as a single injection or continous infusion and can provide analgesia superior than systemic opioids and greater patient satisfaction.

OPIOIDS [20,21,22]:

The term opiate was originally used to refer to drugs derived from opium, including morphine, its semisynthetic derivatives, and codeine. The more general term opioid was introduced to designate all drugs, both natural and synthetic, with morphine like property, including endogenous opioids.

ENDOGENOUS OPIOIDS AND OPIATE RECEPTORS [23,24]:

All of the endogenous opioids are derived from three prohormones: proenkephalin, prodynorphin and pro-opiomelanocortin each encoded by a separate gene. It was in 1970, a number of peptides isolated from brain, spinal cord, plasma, GIT and placenta also had affinity for opiate receptors and had morphine like action. It was hypothesized that these peptides constitute an endogenous opiate system which normally modulate pain

perception, mood, hedonic behavior, motor behavior, pituitary hormone release, GIT motility, diuresis and modulation of respiratory responses to stimuli and drugs. These endogenous opioids are active in very small amounts and their action can be blocked by naloxone. Endogenous opioids are classified into three different families each derived form an unique large precursor polypeptide as mentioned above.

ENDORPHINS: Most important in this family is the beta-endorphin, which has 31 amino acids. Its presence in spinal cord is debatable. Outside the CNS, beta-endorphins exists in GIT, placenta and plasma.

ENKEPHALINS: Most important ones are methionine-enkephaline(met-ENK) and leucine-enkephalin(leu-ENK), which are penta peptides. They have a wide distribution in the CNS such as the amygdala, globus pallidus, striatum, hypothalamus, thalamus, brain stem, dorsal horn. They have also been isolated from PNS such as the peripheral ganglia, adrenal medulla. They have been found to modulate analgesia through the modulation of substance P from the dorsal horn.

DYNORPHINS:

They primarily function as a neuromodulator and are found in the hypothalamo- neurohypophyseal axis. Dynorphins are also found to be distributed in other areas of CNS relevant to nociception like the

periaqueductal grey matter, limbic system, thalamus and dorsal horn of spinal cord. They are found to play a more important role for nociception at the level of spinal cord than in the brain.

OPIATE RECEPTORS [23,24]:

Opiate receptors are located in many areas of the CNS including the substansia gelatinosa of the caudal spinal trigeminal nucleus which carry pain fibres from face and hands via the cranial nerves 5,7,9 and 10.Within the spinal cord, opiate receptors have been demonstrated in the grey matter. The distribution of opiate receptors does not parallel the distribution of the endogenous receptors. The "C" fibre terminals have opiate receptors, but not the "A" fibres. Opiate receptors have also been demonstrated on the presynaptic sites on the dendrites of the second order neurons receiving "A delta" and "C" fibres.

The pharmacological action of morphine and other opioids is mediated by their interaction with these opioid receptors. Radio-ligand binding studies have divided these opioid receptors into four categories: mu, kappa, delta and sigma. Each have a specific pharmacological profile.

Mu(morphine) receptor:

This receptor is named after the drug morphine,based on its binding affinity. It also exhibits high affinity to pethidine, fentanyl and its congeners. It is located both in the brain and spinal cord.The highest concentration have been demonstrated in the periaqueductal gray region, thalamus,substansia gelatinosa, nucleus ambgious and nucleus tractus solitarius. Two sub types have been described: μ1 and μ2 mediating supra spinal analgesia and spinal analgesia respectively.

Kappa(ketocyclazocine) receptor:

This receptor in named after its high affinity to ketocyclazocine. Two sub types: k1 and k3 are clinically important, mediating spinal and supra spinal analgesia respectively. Activation of kappa receptor causes mild to moderate analgesia, ceiling respiratory depression, dysphoria, hallucination, miosis, sedation and physical dependence.

Delta receptor:

This receptor has affinity towards enkephalins and is present in both spinal cord(dorsal horn) and brain(limbic area). Activation of the receptor causes analgesia, respiratory depression, affective behavior, reduced GI motility. Delta receptors mediate spinal analgesia predominantly but the

affective component is mediated through supra spinal receptors in the limbus. The proconvulsant action is more prominent in delta receptor agonists.

Sigma receptor:

It is no longer considered as opioid receptor because this receptor is neither activated by morphine nor blocked by naloxone. However certain drugs like pentazocine and butorphanol have been found to bind to sigma receptor.

PHARMOCOLOGY OF DRUGS

BUTORPHANOL [25,26,27]

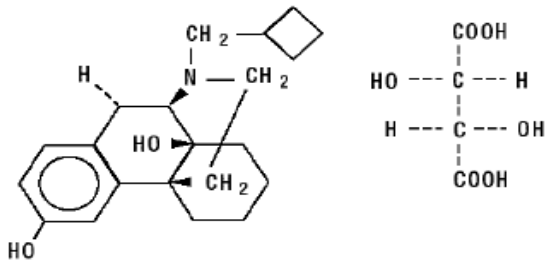
Butorphanol is a synthetic opioid agonist-antagonist, whose actions are found te be mediated through mu, kappa, and sigma receptors. It was found to have significant analgesic property, narcotic antagonistic property, reversibility with naloxone by Montonic in 1973, by studying the drug effect in animals and man.

CHEMICAL NATURE:

Butorphanol is a 3,14-dihydroxy morphinion, with nitrogen substitution. It is a synthetic compound of phenanthrene series existing physically as a water soluble crystal. One milligram molecular weight of salt is equivalent to 0.68mg of free base.

CHEMICAL STRUCTURE AND FORMULA:

$C_{12}H_{29}NO_2C_4H_2O_6$ (FIG 9)



CHEMICAL NAME: (-)17-(CLOBUTYLMETHYL) MORPHINAN-3,14 –DIOL[S-(R*R*)]-2-3 DIHYDROXY IBUTANEDIOATE (1:1) (SALT)

PHARMACODYNAMICS:

MECHANISM OF ACTION:

Butorphanol being a mixed narcotic agonist and antagonist, acts as a partial agonist or weak antagonist at mu receptor and strong agnoist at kappa receptor. Activation of these receptors causes both supra spinal and spinal analgesia, sedation, dysphoria with low abuse potential.

Other effects of butorphanol on CNS include respiratory depression, suppression of cough, stimulation of emetic centre, miosis and sedation. Other effects are alteration in the cardiovascular resistance and capacitance, bronchomotor tone, GI motility and bladder sphincter activity.

POTENCY:

The analgesic potency of butorphanol can be compared with the gold standard opioid analgesic morphine.

TABLE 3: POTENCY OF BUTORPHANOL

DRUG	AGONISTIC POTENCTY BUTORPHANOL:OTHER OPIOID	ANTAGONISTIC POTENCY BUTORPHANOL:OTHER OPIOID
PENTAZOCINE	20:1	10-30:1
MORPHINE	5:1	
PETHIDINE	30-40:1	

EQUIPOTENT ANALGESIC EFFECT:

BUTORPHANOL 2mg = 10mg morphine = 40mg pentazocine = 80mg pethidine Following intra muscular injection of butorphanol, rapid absorption occurs and peak plama levels are attained by 20-40minutes. After nasal administration mean peak blood levels of 0.9-1.4 microgram/ml is attained by 30-60minutes.

PHARMACOLOGICAL EFFECTS:

- Butorphanol blocks pain impulses at specific sites in brain and spinal cord.
- Sedation with butorphanol is commonly noted at doses of 0.5mg and above.
- Butorphanol in doses of about 10-12mg iv over 15mins produces narcosis.
- Because of its affinity to kappa receptor, butorphanol produces unpleasant psychomimetic effects in some individuals.
- Butorphanol in doses of 1mg and above through any route produces nausea and vomiting.
- In individuals with significant respiratory dysfunction, 2mg of butorphanol intravenously causes respiratory depression to a degree comparable to morphine. At higher doses, the magnitude of respiratory depression

doesn't increase due to ceiling effect,but the duration is prolonged significantly.

- Subanalgesic doses exhibit antitussive effects in animal studies.
- Effects on CVS: Analgesic doses of butorphanol in patients with cardiac dysfunction is found to increase pulmonary artery pressure, left ventricular end diastolic pressure(LVEDP), thereby causing an increase in cardiac workload. At higher doses, it causes increase in HR and BP due to increase in endogenous plasma catecholamines. So the use of butorphanol is not recommended in the presence of congestive heart failure and myocardial dysfunction.

PHARMACOKINETICS:

Butorphanol is metabolized in liver extensively, which is qualitatively and quantitatively similar by intravenous ,intra muscular or nasal administration. Butorphanol shows extensive first pass metabolism and its oral bioavailability is 5-17% and so no oral formulation is available.

Hydroxyl butorphanol is the major metabolite of butorphanol, while norbutorphanol in small amounts is also produced.Both of these metabolites have been detected in plasma following administration of butorphanol, with norbutorphanol in trace amounts. Elimination half life of

hydroxybutorphanol is 18hrs, therefore there is considerable drug accumulation when dosing is done to achieve a steady state concentration. Elimination occurs by faeces and urine. When H_3 labelled butorphanol is administered to normal subjects 70-80% of drug metabolite is recovered in urine, where 5% is recovered as butorphanol;40% is eliminated in the urine as hydroxybutorphanol and less than 5% is eliminated as norbutorphanol. The elimination half life of butorphanol is increased in elderly. In patients with creatinine clearance less than 30ml/min, the elimination half life is approximately double as compared to 5-8hrs in normal subjects.

Serum protein binding of butorphanol is 80%, volume of distribution is 305-901L and total body clearance is 52-154L/hour.

THERAPEUTIC USES:

Butorphanol is used:

- As a preanesthetic medication
- To supplement balanced anaestheisa
- Transnasally for migraine
- For post operative analgesia

PRECAUTIONS & CONTRANDICATIONS:

Butorphanol should be used with caution in:

- Patients with respiratory dysfunction-COPD, Bronchial asthma
- Patients with hepatic and renal impairment
- Patients with increased CSF pressure and head injury
- Patients with cardiac disease
- Opioid dependant patients

DRUG INTERACTIONS:

Butorphanol when used along with CNS depressants like alcohol,barbiturates,tranquilizers, antihistamines, may cause prolonged CNS depression. When used, lowest possible dose titrated to effect should be given and frequency of dosing should be reduced. Currently its interaction with monoamino oxidase (MAO) inhibitors is reviewed.

SIDE EFFECTS:

The side effects of butorphanol are:

- Drowsiness, confusion
- Hallucinations, dysphoria
- Hypotension
- Nausea, vomiting

- Respiratory depression
- Urinary retention
- Pruritis
- Hypersensitivity
- Tolerance

FENTANYL [28]

Fentanyl was first synthesized by Dr. Paul Janssen in 1960. It is a μ receptor agonist approximately 100 times more potent than morphine. It is a highly lipid soluble phenylpiperidine derivative and a synthetic opioid with low molecular weight. It is a safe and rapidly acting opioid available in parenteral and also in transdermal , aerosolized formulations.

STRUCTURAL FORMULA:

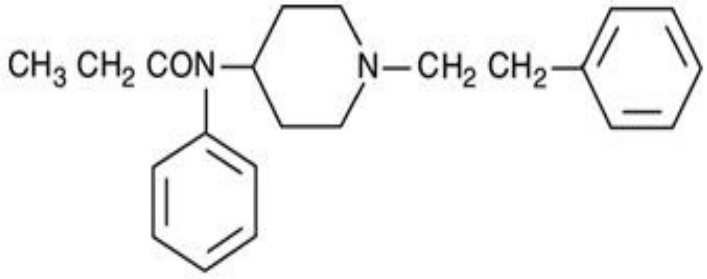


FIG 10: STRUCTURAL FORMULA OF FENTANYL

CHEMICAL FORMULA: $C_{22}H_{28}N_2O.C_6H_6O_7$

PHYSICAL PROPERTIES:

Fentanyl is available as a sterile solution of fentanyl citrate in water for injection(2ml ampoule containing 100mcg of fentanyl). It is clear with pH 4-7.5, pka 8.4 and should be stored at a temperature of less than 40 degree celsius.

MECHANISM OF ACTION:

Primarily a μ receptor agonist, fentanyl has an analgesic potency greater than morphine, pethidine and alfentanyl. Its principle site of interaction is the μ receptors located at the supraspinal sites. Fentanyl also binds with the kappa receptors in the spinal cord to a much lesser degree.

PHARMACOLOGICAL EFFECTS:

***1.Cardiovascular system*:**

a.Heart rate:

Fentanyl causes decrease in heart rate, due to stimulation of central vagal nucleus. Its action is dependant on dose and speed of injection. Fentanyl is a potent stress response attenuator and acts by decreasing the central sympathetic outflow.

b. Cardiac electrophysiological effects:

Fentanyl slows AV conduction, prolongs R-R interval, AV node refractory period and duration of purkinje fibres action potential.

c. Blood pressure:

Centrally mediated reduction in sympathetic vasoregulatory flow, decreases systemic vascular resistance, causing minor reductions in blood pressure ,often associated with bradycardia.

2.Respiratory system:

Fentanyl causes dose dependant respiratory depression. Respiratory rate,tidal volume and resting minute voulume are decreased. Fentanyl blunts the response to hypoxia and hypercapnia.

3.CNS:

Fentanyl produces no change or modest reductions in cerebral blood flow and cerebral oxygen consumption.

5.GIT:

Fentanyl decreases the intestinal motility, causing constipation. It can increase the tone of sphincter of Oddi , there by increasing the pressure in biliary tract producing pain.

6.Muscle tone:

Fentanyl increases the muscle tone, causing rigidity which frequently occurs following iv induction with larger dose. But neuraxial administration of fentanyl is not associated with such effects.

PHARMACOKINETICS:

Intravenous administration of a single dose of fentanyl has a faster onset and shorter duration of action than morphine. Although the onset is rapid there exists a a time lag between peak plasma concentration and peak slowing of EEG. This is because of a delay in effect site equilibration time between blood and brain for fentanyl which is 6.4minutes. Greater lipid solubility enhances passage across blood brain barrier causing a greater potency and onset of action. Fentanyl has a rapid redistribution to inactive tissues like fat, skeletal muscle with associated decrease in plasma concentration, contributing to shorter duration of action. Fentanyl undergoes first pass pulmonary uptake, therby lungs acting as an inactive storage site.

METABOLISM:

Fentanyl undergoes extensive first pass metabolism by N-demethylation producing norfentanyl, hydroxylproprionyl fentanyl and hydroxyproprionyl norfentanyl. Norfentanyl is structurally similar to normeperidine and the principle metabolite in humans. It is excreted in kidneys and can be detected in urine upto 72hrs following a single dose.

ELIMINATION ½ LIFE AND CONTEXT SENSITIVE ½ LIFE:

Elimination half life is 3.1- 6.6hours, although its duration of action is short. This is due to its larger volume of distribution. As the duration of continuous infusion of fentanyl increases beyond 24hours, the context sensitive half life increases. It is 200minutes after 4hours of stoppage of infusion. This is due to saturation of inactive tissues and return of drug to plasma from peripheral compartment.

CLINICAL USES:

Fentanyl is used,

- In the prevention of surgery induced stress response
- In labour analgesia
- As a sole agent for surgical anesthesia

- As transdermal patch preoperatively to allay anxiety and facilitate induction of anesthesia.
- In post operative pain relief.

It is used clinically in a wide range of doses and through different routes. Analgesic dose of fentanyl is 1-2mcg/kg iv. In doses of 2-20mcg/kg, fentanyl has been administered as iv adjuvants to inhalational anesthetics as an attempt to blunt intubation response. Large doses of fentanyl has been used as sole anesthetic because of its minimal effect on CVS, lack of histamine release and potent stress response suppression. Intrathecal/epidural administration of fentanyl produces rapid and profound analgesia.

CONTRAINDICATIONS:

- Patients on MAO inhibitors within previous 24hrs

SIDE EFFECTS:

The side effects of fentanyl include:

- Bradycardia
- Hypotension
- Pruritis
- Urinary retention
- Respiratory depression

- Occular dysfunction
- Sexual dysfunction
- Hypersensitivity and anaphylaxis.

REVIEW OF LITERATURE

The discovery of spinal opioid receptors and the subsequent development of the technique of epidural and intrathecal opioid administration is undoubtedly, one of the most significant advances in pain management in the last three decades. Spinal opioids can provide profound postoperative analgesia with fewer central and systemic adverse effects than with opioids administered systemically.

Opiate receptors were identified in the CNS in 1973 by Pert and Synder and in 1977 large populations of these receptors were localized in the dorsal horn of the spinal cord. These observations, coupled with the discovery of endogenous opioids, led to animal studies that demonstrated that intrathecal administration of opioids produces analgesia[29].

Resill SI et al., in 1976 has described Visual Analogue Scale which is the easiest way to measure the intensity of pain.It is simple to use, efficient and can be analyzed quickly. It is a very sensitive way to assess intensity of pain. VAS can measure efficiency of analgesia by a particular analgesic by noting the scores before and after treatment, but the main drawback of VAS is that it assumes pain to be one-dimensional and

measures only the intensity of pain, whereas the nature, location and psychosocial aspect of pain are not taken into consideration[30] .

Behar in 1979 first reported the effective use of epidural opioids in humans. This initial study provided evidence that opioids reached the spinal fluid very rapidly and that analgesia could be obtained in the absence of "analgesic blood concentrations". Since other neurological functions, including sympathetic vasoconstrictor responses were intact, the term 'selective spinal analgesia' was suggested [31].

In 1979, Ameer B, Slater FJ reviewed chemistry, pharmacology, uses, side effects, pharmacokinetics and dosage of butorphanol tartrate. When administered IM or IV, butorphanol tartrate appears to be as effective for relieving moderate to severe pain as are morphine, meperidine and pentazocine. Butorphanol produces sedation more commonly and at therapeutic dosages, depresses respiration as much as other narcotic analgesics. A limited number of long term clinical studies suggest a lower physical dependence liability with butorphanol than with other narcotic analgesics. Butorphanol's cardiovascular effects are not completely understood, so morphine remains the drug of choice for pain associated with myocardial infarction [32].

Naulty MD et al., in 1984 in Harvard Medical School, Boston, had conducted a double blind randomized dose response study of epidural butorphanol in post caesarean delivery patients, using 1, 2, 4 or 6mg of butorphanol tartarate. After the injection, subjects were evaluated at 5, 10, 15 and 30 minutes and then at 30 minutes intervals for at least 5 hours following the injection. The onset of sensory analgesia was defined as the time elapsed when the patients had no sensory blockade detectable by pinprick and the duration of analgesia was defined as the time elapsed when the patient experienced any pain (a pain score >0 on the linear VAS). A statistically significant ($p< 0.01$) prolongation of postoperative analgesia was seen withbutorphanol doses greater than 1 mg and increasing duration of analgesia seen with increasing dose. Finally, increasing doses of epidural butorphanol significantly decreased the amount of narcotic required in the first 24hrs. Somnolence (easily arousable with a quiet verbal stimulus) of mean duration of approximately 6hrs was the only significant side effect encountered. No patients reported pruritis, nausea, dysphoria or respiratory depression during the course of study [33].

Naulty JS et al., in 1985 evaluated suitability of fentanyl for epidural use and the dosages required in the parturient in a double-blind, randomized manner in 30 ASA 1 patients following caesarean delivery. The patients (5 each group) were randomly assigned to receive 0, 12.5,

25, 50, 70, or 100 µg of fentanyl through the epidural catheter. Level of sensory block, motor block, and pain intensity was assessed. 50 µg of fentanyl produced pain scores of 0 within 9 min and 100 µg in 3-6 min. With Fentanyl 25 µg, mean duration of analgesia was 3 hrs and higher doses produced an increase of approximately 1.5 times [34].

Mok et al., in 1986 evaluated the analgesic efficacy and safety of epidural butorphanol in comparison to epidural morphine, in patients with postoperative pain, in a double blind controlled design. Postoperatively patients were divided into 2 groups in a randomized, double- blind fashion. Group A patients received, epidural butorphanol 4 mg in 10 ml normal saline and patients in Group B received morphine 5 mg in 10 ml normal saline epidurally.The onset of pain relief with epidural butorphanol appeared at 15 minutes, peaked at 30 minutes and duration of action averaged 5.4 hours; whereas with epidural morphine onset of analgesia appeared at 25 minutes, with a peak effect at 1 hour and duration of action averaged 15.2 hours.This study concluded that epidural butorphanol appears to provide efficacious pain relief without much untoward effects in patients with post surgical pain [35].

WE Ackerman in 1988 in his study evaluated and compared the duration of analgesia and side effects of equipotent doses of fentanyl (a pure agonist), buprenorphine (an agonist - antagonist) and butorphanol (an agonist – antagonist).He used 50µg of fentanyl, 0.3mg of buprenorphine and 2mg butorphanol epidurally for comparison in postoperative pain relief. The duration of analgesia was defined to be from the time of epidural study solution injection until the patient experienced any pain (a score > 0 on a 0-10 verbal response scale). The duration of buprenorphine was longer (388.06 ± 54.6 min.) and that of butorphanol is (117 ± 36.4 min.).Sedation incidence was higher with butorphanol and no nausea / vomiting, disturbances in micturation or respiratory depression were noted in any group [36].

Maurice Lippmann in 1988 has reported a study utilizing epidural butorphanol 4mg for pain relief in non-obstetric patients after abdominal operations and showed that epidural butorphanol provided pain relief with rapid onset (15 minutes) but substantially shorter duration (5.6 hrs) than that of epidural morphine 5mg. With serial blood gas measurements, epidural butorphanol 4mg did produce statistically significant elevation in Paco2 even though no clinically significant respiratory depression (RR < 10 breaths / min) was observed in these patients. They concluded that epidural butorphanol might be an interesting alternative to epidural morphine [37].

Catherine O Hunt et al., in 1989 used increasing doses of epidural butorphanol (1,2,3 mg) along with local anaesthetics (0.25% bupivacaine) in 42 term multiparous patients for labour analgesia administered during 1st stage of labour when cervix was 5cm dilated and had minimum VAS pain score. The time from injection of the drug to complete pain relief (VAS – 0) was shortened by addition of butorphanol (21.3 ± 52 min in plain bupivacaine group, 16.5 ± 3.6 min with 1mg butorphanol, 6.9 ± 3.6min with 2mg and 6.3 ± 3.7 min with 3mg of butorphanol). The duration of complete pain relief was prolonged by addition of butorphanol. In the groups in which 2 and 3 mg butorphanol was used, the total dose of bupivacaine used was reduced by more than 50% in total requirement ($p < 0.01$) than that used in the control group. The incidence of somnolence increased with higher dose of butorphanol. No patients had nausea, hypotension and respiratory depression. Two patients who received butorphanol 2mg early in labour complained of dysphoria (a recognized side effect of Kappa-opiate receptor agonist, more common with pentazocine) [38].

Table 2: Catherine O Hunt Study

Dose of butorphanol	**Duration of Analgesia**
No butorphanol	59 ± 12.3 min.
1mg butorphanol	67 ± 15.2 min.
2mg butorphanol	137 ± 18.4 min.
3mg butorphanol	220 ± 24.1 min

Ackerman in 1989 compared the incidence of pruritis after epidural administration of opioid in a controlled study of post cesarean section population, the incidence of pruritis was highest among patient receiving morphine, second highest with epidural fentanyl.

Table 3: Ackerman Study [36]

Opioid	**Dose**	**Incidence of pruritis**		**Duration of analgesia (min)**
		n (15)	%	
Morphine	5mg	9	60	932 ± 87
Fentanyl	50µg	7	47	145 ± 38
Buprenorphine	0.3mg	0	0	388 ± 55
Butorphanol	1mg	1	7	97± 23

Salomaki TE et al., in 1991 compared epidural and intravenous fentanyl infusions for pain relief for the first 20 hrs after thoracotomy in 40 patients, who were assigned randomly to receive either fentanyl epidurally and saline intravenously or fentanyl intravenously and saline epidurally in a double blind fashion. Patients reported similar median pain scores, but in the epidural group the required mean fentanyl infusion rate, plasma fentanyl concentrations, incidence of nausea and sedation were less and respiratory functions were better preserved. Thus they found that there is clinical advantage of the epidural infusion over the intravenous infusion of fentanyl [39].

Lytle SA et al., in 1991 did a retrospective analysis of 133 patients who received fentanyl for postoperative analgesia 5µg/ml as continuous epidural infusions. 59.3% of the patients did not receive any additional narcotics but 26.3% did.The side effects were less and respiratory depression did not occur. Urinary retention occurred in one patient, pruritis in 4% and nausea in 25.5%. They shown that epidural fentanyl provides good to excellent pain relief with minimal side effects [40].

Quisqueya T et al., in 1991 compared epidural butorphanol (1, 2 and 4mg) and morphine (5mg) for post caesarean section analgesia. Each dose of butorphanol produced greater pain relief than morphine at 15, 30, 45 and 60min ($p<0.05$). At 90 min and two hours each dose of butorphanol and morphine produced similar pain relief. The time of onset of epidural

analgesia following butorphanol was more rapid than following morphine and 14, 22 and 17% of patients treated with butorphanol 1, 2 and 4mg respectively had not requested supplemental medications at eight hours. They concluded that epidural butorphanol is safe and effective in providing postoperative analgesia and may be particularly useful in clinical situations where,

1. A prompt onset and / or limited duration of analgesic action may be indicated.

2. Epidural patient – controlled Analgesia (PCA) is used.[41]

In 1991, Palocios et al., compared epidural butorphanol 1, 2, and 4mg with epidural morphine 5mg for postoperative analgesia in 92 healthy term parturients who had undergone caesarean section under epidural anaesthesia in a randomized double blind study. Postoperative pain was assessed using a visual analog scale and recorded with heart rate, blood pressure and respiratory rate. The mean pain scores at 15, 30, 45, and 60 minutes following each dose of butorphanol were significantly lower than corresponding values in the patients who received morphine.The time of onset of epidural analgesia following butorphanol was more rapid than following morphine. Pruritis occurred in only 1.4 percent of butorphanol

patients compared with 43 percent of the morphine patients. Thus the study concluded that epidural butorphanol is safe and effective in providing epidural analgesia [42].

Kim DH, Kim TJ, Park NH in 2002 studied the efficacy of pain relief, adequate infusion dosage and the side effects of epidural butorphanol and compared with those of epidural fentanyl.

In this study forty consenting, healthy, term parturients who had undergone Caesarean section under epidural lidocaine anaesthesia received 0.1% bupivacaine/ butorphanol 50 µg/ml (Group 1, n=20) or 0.1% bupivacaine/fentanyl 5 µg/ml (Group 2, n=20) using patient controlled epidural analgesia (PCEA), were evaluated. RESULTS:The total amount of 48hr consumption was 19.4 mg (butorphanol) and 1546.8 µg (fentanyl). There were no significant differences between the two groups in total infusion doses of the above drugs, VAS score and side effects. The potency ratio of fentanyl/butorphanol was 1/12.5. They concluded that both butorphanol and fentanyl were useful and safe drugs for PCEA for postoperative pain control and a combination of butorphanol and bupivacaine provided more economically effective analgesia.

Hwang KB, Chung CJ, Lee SC et al., in 2004 observed that epidural butorphanol produces effective analgesia with fewer side effects than morphine in obstetric patients. The analgesic efficacy and side effects of epidural butorphanol were compared with epidural fentanyl for patient controlled epidural analgesia (PCEA) after gastrectomy. After obtaining patient consent, 75 patients undergoing elective gastrectomy were randomly allocated to epidural butorphanol and epidural fentanyl groups. An epidural catheter was introduced at the T7-8 or T9-10 intervertebral spaces before operation. Postoperative analgesia was provided using a PCEA with either fentanyl 5µg/ml or butorphanol 50µg/ml in a 0.05% bupivacaine solution. The PCEA was set to deliver a bolus of 2 ml, with a lockout interval of 10 min and no basal infusion. PCEA consumption, pain intensity using a visual analog score (VAS), patient's satisfaction to PCEA and side effects were evaluated. No significant difference in PCEA consumption, VAS pain score, or patient's satisfaction to PCEA was found between the two groups. The incidence of pruritis in the butorphanol group was less than that in the fentanyl group ($P < 0.05$). The study concluded that PCEA with butorphanol provided similar postoperative analgesia with less pruritis than fentanyl in patients undergoing gastrectomy.

Premila Malik, Chhavi Manchanda, Naveen Malhotra in 2006 conducted a study to assess and compare the safety and efficacy of postoperative analgesia with epidural butorphanol and fentanyl. The prospective, randomized double blind study was conducted in 60 patients, aged 20-60 years, belonging to ASA physical status class I or II and scheduled for elective orthopaedic hip or lower limb surgery to assess the safety and efficacy of postoperative analgesia with epidural butorphanol compared with epidural fentanyl. All the patients were administered combined spinal epidural anaesthesia. Patients were randomly allocated to two groups: Group A (n=30) - epidural butorphanol (2 mg) followed by top up doses of 0.5 mg was administered for postoperative analgesia and Group B (n=30) - epidural fentanyl (50 μg) followed by top up doses of 15 μg was administered for postoperative analgesia.The timing of incremental doses, interval between injections and total dose of analgesic drug given in 24 hours were recorded. There were no significant changes in pulse rate, systolic blood pressure, diastolic blood pressure, respiratory rate and peripheral arterial oxygen saturation in the two groups at different time intervals throughout the 24- hours study period ($p>0.05$).The duration of postoperative analgesia was longer and pain relief clinically better in butorphanol group. The sedation scores remained significantly higher in butorphanol group at all postoperative time intervals over 24 hours

($p<0.05$). The incidence of nausea and pruritis was higher in epidural fentanyl group ($p<0.05$). They concluded that both butorphanol and fentanyl are effective and safe drugs for postoperative epidural analgesia with minor side effects [44].

Gough JD et al., in 1988 in their study for postoperative pain relief tried a loading dose of epidural fentanyl of 1.5 µg/kg in 10 ml of sterile solution followed by continuous infusion. Range of mean (SD) of cardio-respiratory variables were found to be 84(2)-95 (18) beats/min for H.R., 121 (19)-133(14) mm Hg for systolic arterial pressure, 70(10)- 76 (10) mm Hg for diastolic arterial pressure and 21 (3)-23 (4) / min for respiratory rate.Their study concluded that 1.5µg/kg of fentanyl provided better hemodynamic stability in the postoperative period .

MATERIALS AND METHODS

This study is a comparative and prospective study conducted at Thanjavur Medical College. After obtaining clearance from institutional ethical committee and informed consent, a total of 60 patients of either sex aged between 20-60years belonging to ASA physical status I &II scheduled for elective lower abdominal surgeries were randomly selected.

INCLUSION CRITERIA:

- Patients aged between 20-60yrs
- Weighing between 40-70kgs
- Both male and female
- ASA grade I&II
- Patients undergoing elective lower abdominal surgeries

EXCLUSION CRITERIA:

- Pregnant women
- Patient with h/o cardiac and respiratory disorders
- Patient with h/o hepatic and renal diseases
- Patient with h/o convulsions, neurological deficits
- Spinal deformities and psychiatric diseases
- ASA grade III & IV
- Coagulopathies and patients with infection at the puncture site.

METHODOLOGY

60 patients posted for elective lower abdominal surgeries were randomly selected for the study. All patients were thoroughly examined and investigated preoperatively one day before surgery and explained about the anesthetic technique. Routine preoperative investigations were done. All the patients were educated about the verbal numerical pain scale for assessment of pain.

Grading of post operative pain is done using Visual analog Scale(VAS).The patient will be asked to quantify their pain using VAS pain scale, giving a score of 0 to10, with 0- indicating no pain and 10 indicating the worst possible pain.

Written informed consent was obtained. All patients received premedication at 10p.m on the night before surgery with Tab. Alprazolam 0.25mg and Tab. Ranitidine 150mg and thereafter advised nil per oral.

On the day of surgery patients were shifted to the operating room, and multiparameter monitors were connected. The base line heart rate, SpO_2 and blood pressure(systolic, diastolic and MAP) were recorded. An 18G iv cannula was inserted and patients were preloaded with 10ml/kg of Ringer lactate over 15-30minutes prior to epidural block.

The anaesthesia machine, airway equipments and emergency drugs were kept ready.

Patients were positioned in right lateral decubitus posture. Observing sterile precautions L3-L4 space was identified. Skin was infiltrated with local anesthetic inj. 1% lignocaine 2ml. Epidural space was identified with an 18G Tuohys needle, by using loss of resistance to air technique and a 19G epidural catheter was inserted about 5cms into the epidural space and secured in place. Throughout the procedure patient's vitals were monitored.

` A test dose of 3ml of 1.5% lignocaine with adrenaline(1:2,00,000) was given to rule out intravascular or intrathecal placement of the catheter. The patient was made to lie supine. Five minutes after test dose, confirming the absence of intrathecal or intravascular placement, 20ml of study drug was injected through epidural catheter depending on the study group.

Patients were divided into two groups:

Group BB 30 patients	Bupivacaine with butorphanol group 0.5% bupivacaine (18ml) with 1ml of 1mg butorphanol (preservative free) with 1ml of sterile normal saline to make a total of 20ml.
Group BF 30 patients	Bupivacaine with Fentanyl group 0.5% Bupivacaine 18ml with 2ml(100mcg) of fentanyl (preservative free).

All patients were given oxygen at 5L/min through face mask.

No intravenous analgesics or sedation were administered during the surgery.

The time of injection of study drug was noted at "0" time.The drug was injected approximately at the rate of 1ml/second and the height of sensory blockade was determined by eliciting pin prick test

In the perioperative period the following parameters were studied.

1.Vital parameters such as HR,BP, SPO_2,RR were continuously monitored every 5mins for the first 15mins and then onwards every 15mins throughout the intraoperative period and every ½ an hour in the post

operative period for 2hours. Intraooperative hypotension if any was treated with iv fluids, O_2 supplementation and titrated doses of ephedrine 3-6mg or mephenteramine 3-6mg iv. Bradycardia if any was treated with Inj. Atropine.

2.Onset of analgesia is the time taken from injection of local anaesthetic solution upto loss of pin prick sensation in any dermatome.

3. Completion of analgesia is the time taken from the initial onset of analgesia upto the time when analgesia attained its maxium dermatome level, with no further rise for 5mins.

4. Quality of analgesia was graded as follows:

- Good- No complaint of pain or discomfort during the procedure
- Fair-Pain or discomfort felt only during specific stages of procedure, like traction on viscera/peritoneum.
- Poor-Pain during surgery and needed top up with epidural local anaesthetic solution.

5. Duration of analgesia is the time taken from the onset of analgesia upto the time when VAS reached a score of 5.

6.Sedation score was assessed using subjective sedation score:

0 awake, conscious, no sedation to slightly restless

1 calm and composed

2 awakens on verbal commands

3 awakens on gentle tactile stimulation

4 awakens only on vigorous shaking

5 unarousable

POST OPERATIVE OBSERVATIONS:

The following parameters were observed in the post operative period:

1.Pain score – VISUAL ANALOGUE SCALE, every hour till 8hrs.

2.Vitals were recorded at the same time intervals as the pain score.

When the VAS score reached 5, rescue analgesia was given through the epidural catheter and the study in the patient ceased.Complications like nausea, vomiting, urinary retention,headache, pruritis and respiratory depression if any were noted and treated accordingly.

OBSERVATION AND RESULTS

The data collected was subjected to statistical analysis using Statistical Package for Social Sciences. Chi-square test and the student 't' test was used to test the significance of difference between the two groups. A 'p' value <0.05 was taken to denote a significance.

TABLE 4: COMPARISON OF AGE

Both the groups were comparable with respect to demographic profiles like age, sex, weight.

GROUP	**MEAN(years)**	**S.D±**	**Statistical significance**
BB Group(n=30)	41.37	6.071	T= -1.96 Df = 48 0.8457> 0.05
BF Group(n=30)	41.63	4.311	

Patients aged 20 – 60yrs were included in the study. The mean age is 41.37 years in BB group and 41.63 years in BF group. There is no statistical difference in the age comparison between the two groups.

GRAPH 1: AGE COMPARISON

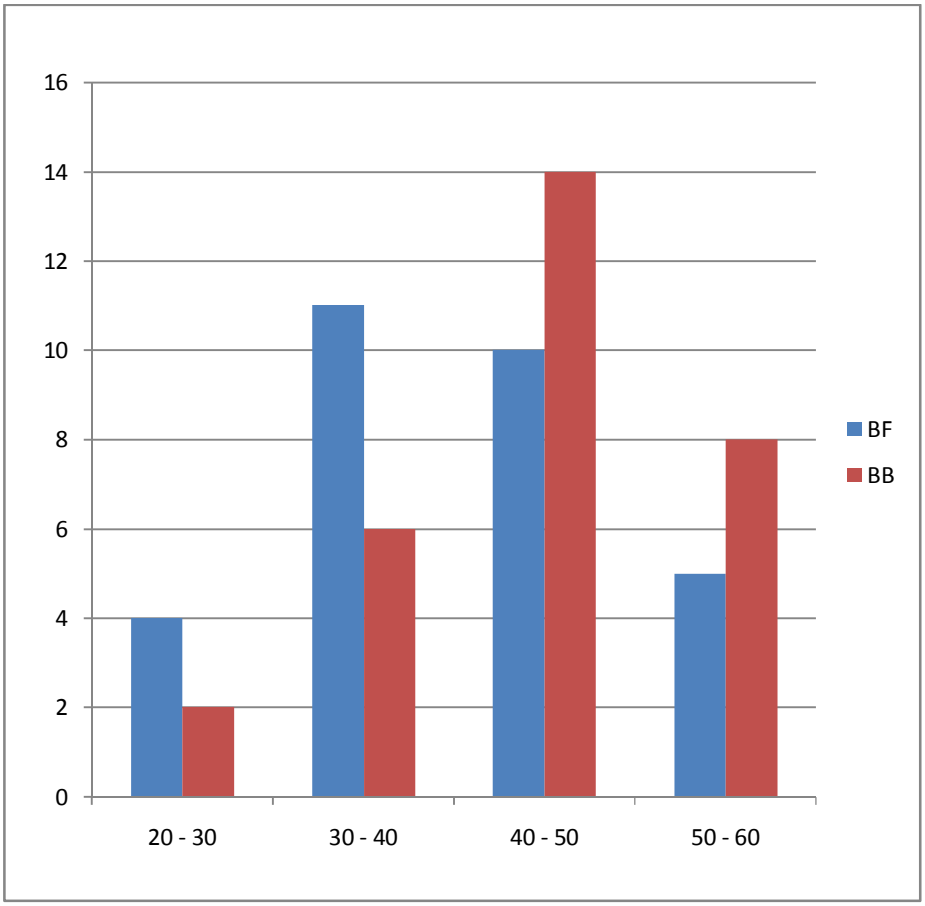
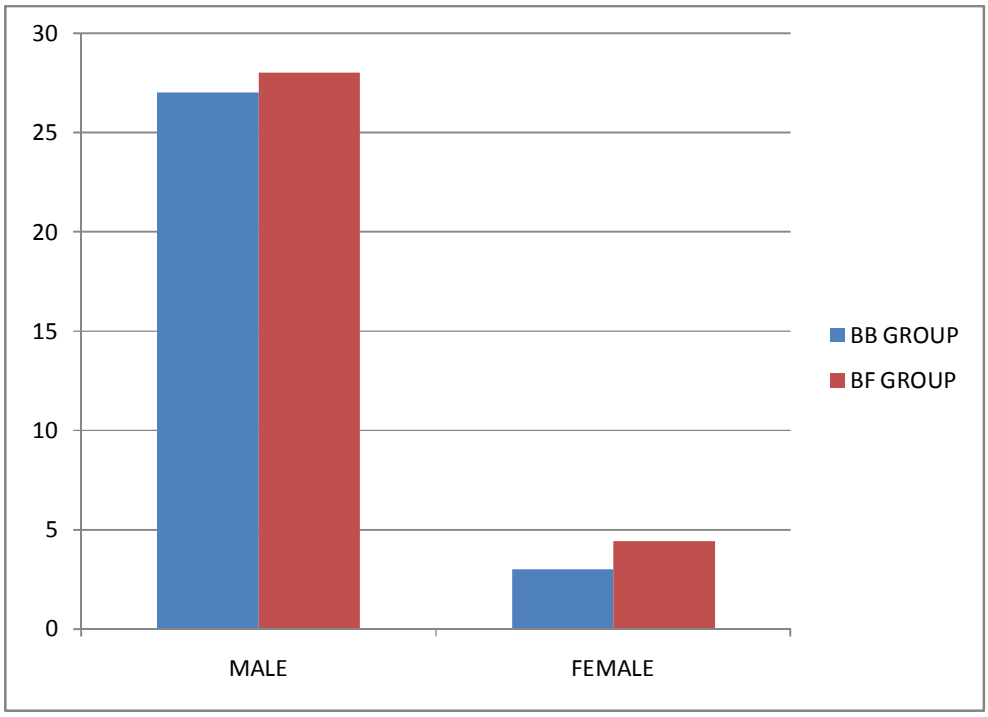


TABLE 5: SEX DISTRIBUTION

SEX	***BB GROUP*** ***N = 30***	***BF GROUP*** ***N = 30***	**Statistical signifiance**
MALE	27(90%)	28(92%)	$X^2 = 0.758$ Df = 1 0.384 > 0.05
FEMALE	3(10%)	2(8%)	

90% of patients in BB group are males, 92% in BF group are males. 10% in BB group are females and 8% in BF group are females. There is no statistical difference in sex comparison between the two groups.

GRAPH 2: SEX COMPARISON



Majority of patients in both study groups are males.

TABLE 6: COMPARISON OF HEART RATE

TIME IN MINUTES	GROUP BB		GROUP BF		STATISTICAL INFERENCE
	MEAN/MINS	S.D	MEAN/MINS	S.D	
0	84.50	±7.408	83.80	±7.392	0.769>0.05 NS
5	87.33	±9.488	87.80	±9.368	0.849>0.05 NS
10	84.87	±9.850	86.17	±8.848	0.593>0.05 NS
15	82.87	±9.726	84.53	±8.693	0.487>0.05 NS
20	80.83	±9.959	83.57	±8.451	0.256>0.05 NS
25	79.97	±9.764	83.13	±8.645	0.189>0.05 NS
30	78.87	±10.06	82.03	±8.282	0.189>0.05 NS
40	77.23	±9.517	81.07	±7.570	0.090>0.05 NS
50	75.60	±9.328	75.68	±9.126	1.000>0.05 NS
60	74.77	±9.175	74.67	±9.089	1.000>0.05 NS
70	74.33	±8.856	74.13	±8.813	1.000>0.05 NS
80	73.97	±9.528	74.17	±9.108	0.935>0.05 NS
90	73.07	±9.303	73.20	±9.103	0.955>0.05 NS
100	72.80	±8.608	73.10	±8.658	1.000>0.05 NS
110	72.43	±8.557	72.32	±8.432	1.000>0.05 NS
120	72.20	±7.490	72.57	±7.398	1.000>0.05 NS

There is stastically no significant difference in mean heart rate from 5 minutes to 120 minutes between the groups BB and BF. Mean heart in BB group was 75.60/min and BF group was 74.67/min.

GRAPH 3: COMPARISON OF MEAN HEART RATE

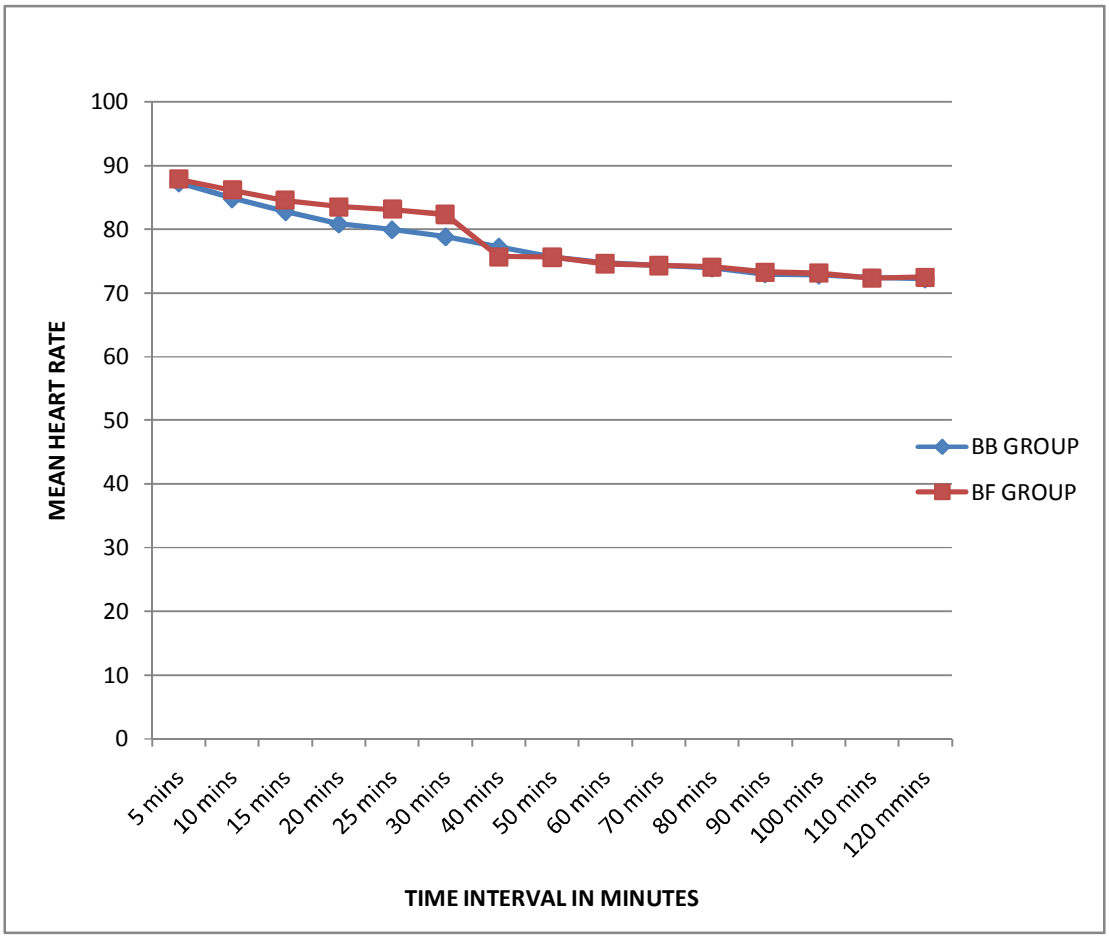


TABLE 7: CHANGES IN MEAN ARTERIAL BLOOD PRESSURE

TIME IN MINUTES	GROUP BB		GROUP BF		STATISTICAL INFERENCE
	MEAN/mmHg	S.D	MEAN/mmHg	S.D	
0	92.7667	±6.083	89.7000	±7.278	0.969>0.05 NS
5	90.8667	±7.152	90.6667	±8.035	0.919>0.05 NS
10	87.6000	±6.975	87.4000	±7.686	0.918>0.05 NS
15	85.2000	±7.274	85.0667	±7.965	0.996>0.05 NS
20	83.4333	±7.219	83.3667	±7.880	0.973>0.05 NS
25	82.1333	±6.489	81.9333	±7.248	0.911>0.05 NS
30	80.5000	±6.323	80.3333	±7.018	0.923>0.05 NS
40	79.6667	±6.171	79.5667	±6.382	0.951>0.05 NS
50	78.8000	±5.695	76 7.7333	±5.976	0.965>0.05 NS
60	79.0000	±5.219	78.9333	±5.426	0.961>0.05 NS
70	80.3000	±4.403	80.3000	±4.587	1.000>0.05 NS
80	81.1667	±4.534	81.3000	±4.617	0.911>0.05 NS
90	83.2333	±4.336	82.6667	±4.412	0.618>0.05 NS
100	84.0667	±4.109	83.5333	±4.116	0.617>0.05 NS
110	86.1000	±4.369	85.4333	±4.272	0.550>0.05 NS
120	87.2333	±4.328	86.4333	±4.264	0.474>0.05 NS

There is statistically no significant difference in the mean arterial pressure from 5 minutes to 120 minutes between BB group and BF group. The mean arterial blood pressure in BB group is 83.4 mmHg±1.26(S.D.) and in BF group is 81.3 mmHg±1.05(S.D.).

GRAPH 4: COMPARISON OF MEAN ARTERIAL BP

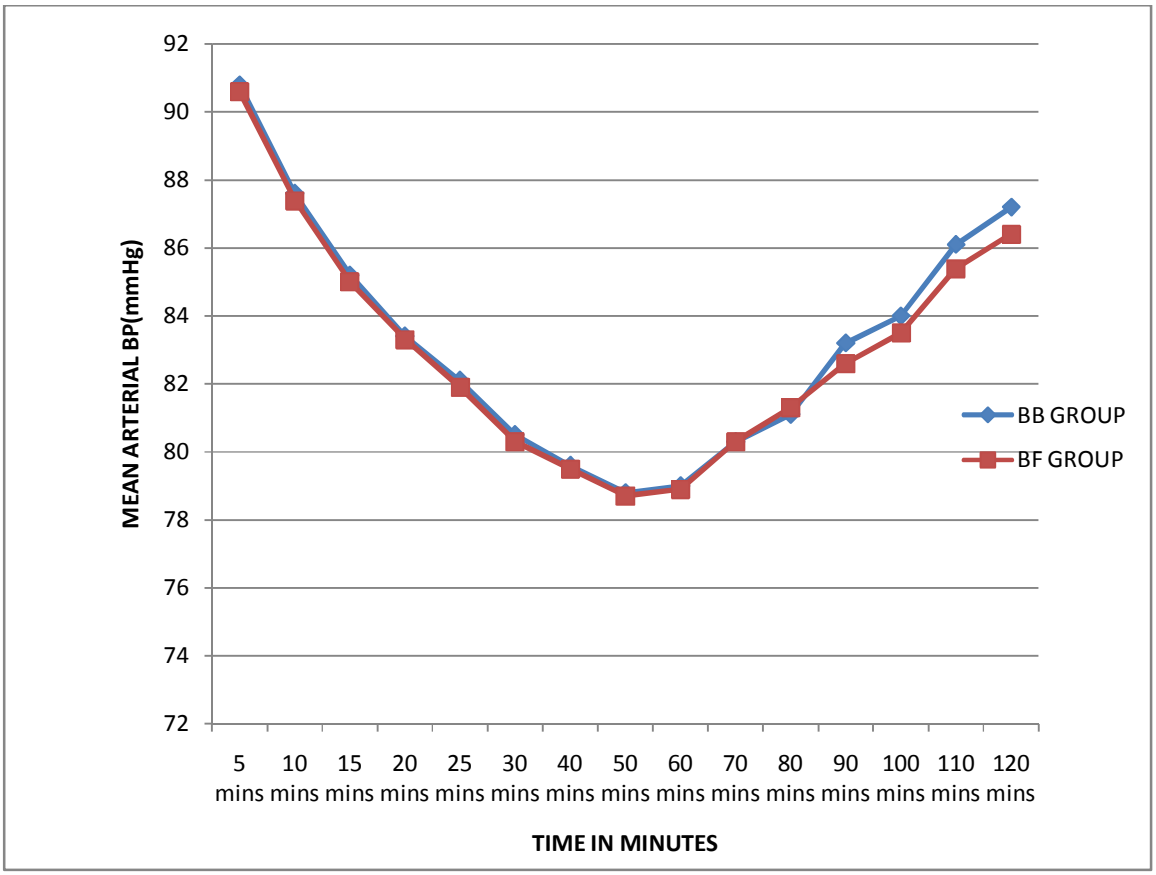


TABLE 7: COMPARISON OF MEAN RESPIRATORY RATE/MINUTE

TIME IN MINUTES	MEAN/MINUTES		S.D. ±		STASTISTICAL INFERENCE
	GROUP BB	GROUP BF	GROUP BB	GROUP BF	
0	13.4	13.7	1.042	0.868	0.286>0.05 NS
5	13.7	13.2	0.069	0.695	1.000>0.05 NS
10	14.9	14.2	1.709	1.092	0.064>0.05 NS
15	14.6	14	1.401	1.050	1.000>0.05 NS
20	13.7	13.8	1.250	0.924	0.967>0.05 NS
25	13	13.4	1.188	1.072	0.176>0.05 NS
30	12.8	12.9	1.156	1.033	0.558>0.05 NS
40	12.2	12.6	1.104	0.932	0.170>0.05 NS
50	12	12.5	1.080	0.937	0.492>0.05 NS
60	12	12.1	1.082	0.791	0.499>0.05 NS
70	11.9	11.83	1.172	0.647	0.684>0.05 NS
80	12.0	12.2	0.932	0.839	1.000>0.05 NS
90	12.1	12	1.074	0.909	0.606>0.05 NS
100	12.3	12.1	0.994	0.937	0.426>0.05 NS
110	12.2	12.4	0.935	0.865	0.392>0.05 NS
120	12.3	12.5	0.927	0.858	0.390>0.05 NS

There is statistically no significant difference in the mean respiratory rate from 5mins to 120mins between BB group and BF group. The mean respiratory rate in BB group is 12.6 and in BF group is 12.9.

GRAPH 5: MEAN RESPIRATORY RATE IN BB AND BF GROUPS

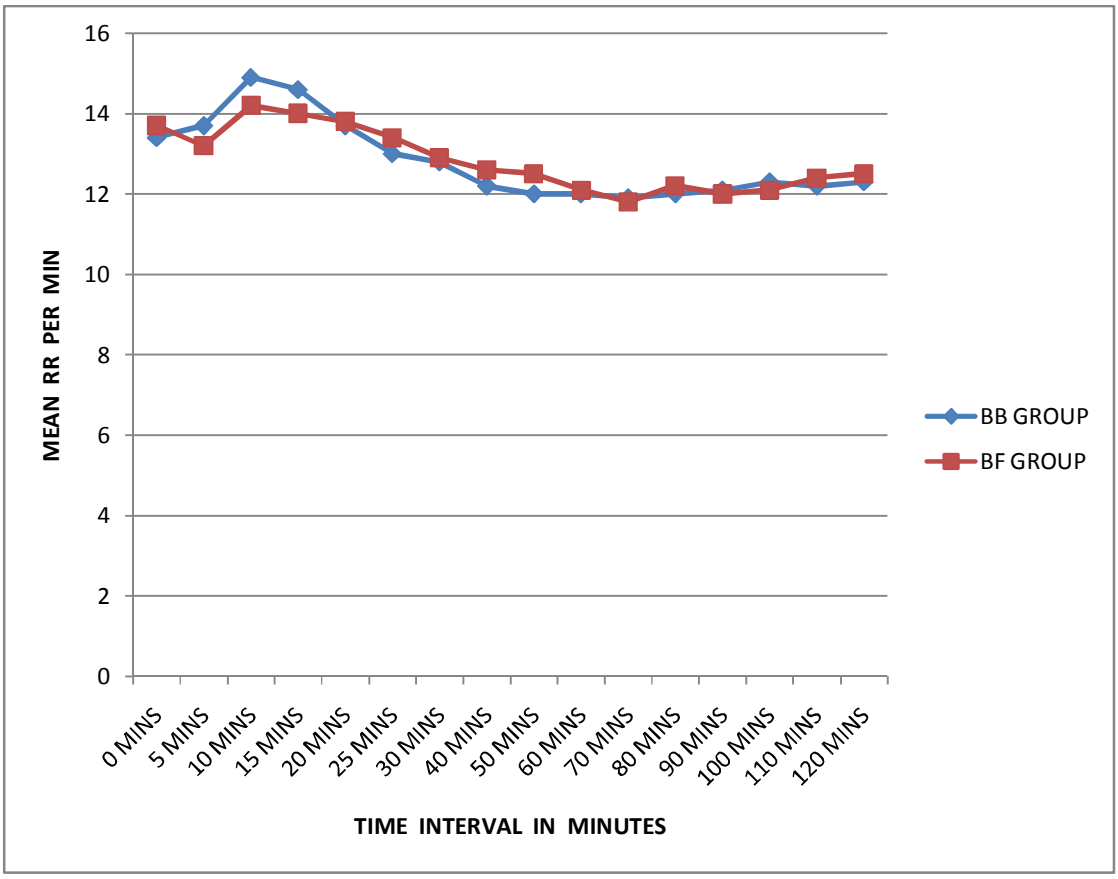


TABLE 8: COMPARISON OF MEAN SPO_2 IN BB AND BF GROUPS

TIME IN MINUTES	GROUP BB		GROUP BF		STASTISTICAL INFERENCE
	MEAN %	S.D.	MEAN %	S.D.	
0	98.5	±0.507	98.5	±0.504	0.799>0.05 NS
5	98.4	±0.502	98.5	±0.501	0.769>0.05 NS
10	98.4	±0.500	98.4	±0.502	0.748>0.05 NS
15	98.3	±0.479	98.3	±0.504	0.434>0.05 NS
20	98.2	±0.450	98.2	±0.479	0.581>0.05 NS
25	98	±0.365	98	±0.450	0.302>0.05 NS
30	98	±0.183	98	±0.365	0.656>0.05 NS
40	98	±0.101	97.9	±0.183	0.321>0.05 NS
50	98	±0.263	97.9	±0.183	0.570>0.05 NS
60	97.7	±0.365	97.8	±0.346	0.477>0.05 NS
70	97.6	±0.485	97.6	±0.498	0.125>0.05 NS
80	97.6	±0.508	97.6	±0.490	0.305>0.05 NS
90	97.8	±0.379	97.7	±0.449	0.356>0.05 NS
100	97.8	±0.345	97.8	±0.345	1.000>0.05 NS
110	97.8	±0.365	97.8	±0,182	0.656>0.05 NS
120	97.9	±0.253	98	±0.101	0.155>0.05 NS

There is no statistical significance in mean Spo_2 from 5 minutes to 120 minutes in between BB group and BF group.

GRAPH 6: TREND OF MEAN SPO_2 IN BB AND BF GROUPS

OVER 0 MINUTES TO 120 MINUTES

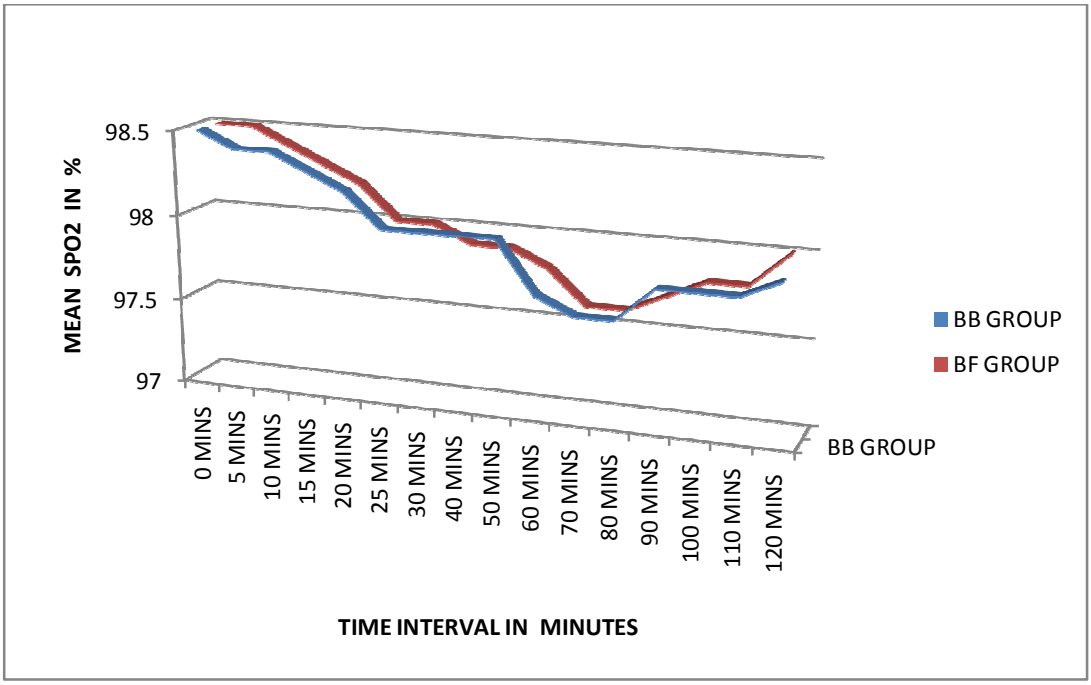


TABLE 9: SEDATION CHARACTERISTICS

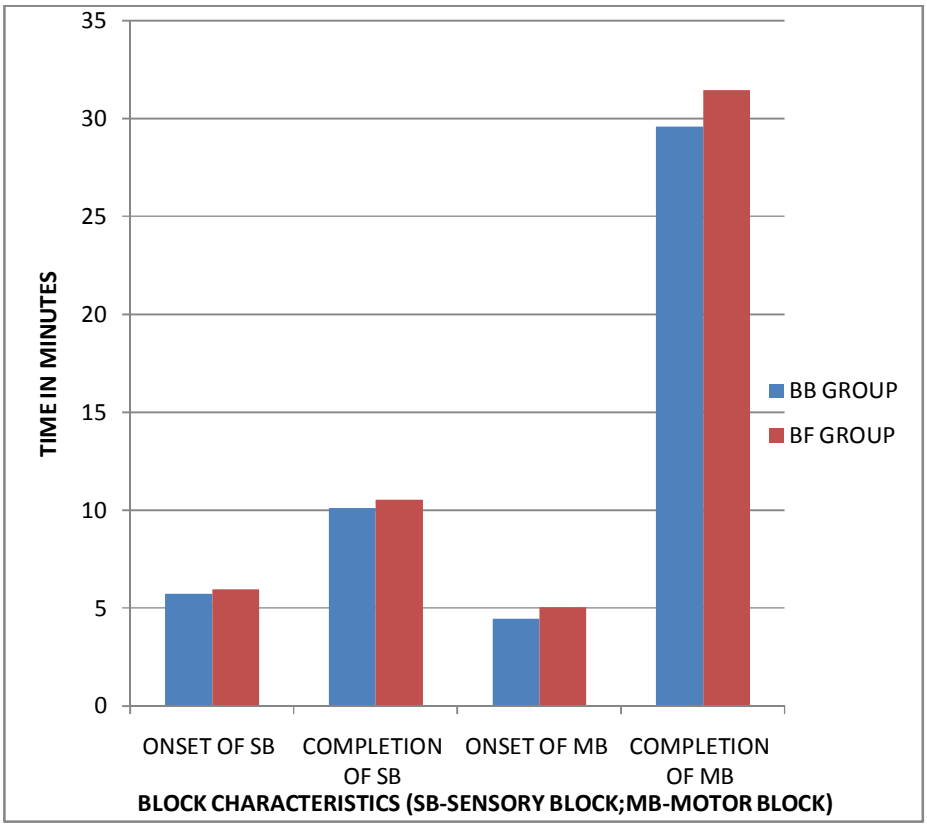
TIME IN MINUTES	SUBJECTIVE SEDATION SCORE(% OF CASES												STATISTCAL INFERENCE
	GROUP BB						GROUP BF						
	0	1	2	3	4	5	0	1	2	3	4	5	
30		100					85	15					0.001< 0.05 S
60			100					100					0.001< 0.05 S
90			100					80	20				0.000< 0.05 S
120			75	25			12	32	56				0.003< 0.05 S

In group BB, 100% patient had sedation score of 1 at 30 minutes, whereas in group BF 85% patients had a sedation score of 0 at 30 minutes. At 60,90and 120 minutes majority of the patients in group BB had a sedation score of 2 and 3, whereas in group BF the sedation score was 1and 2.

TABLE 10: COMPARISON OF ONSET AND COMPLETION OF SENSORY BLOCK IN GROUP BB & BF

PARAMETERS	GROUP BB		GROUP BF		STATISTICAL INFERENCE
	MEAN IN MINUTES	S.D.	MEAN IN MINUTES	S.D.	
ONSET OF SENSORY BLOCK	5.73	1.48	5.96	1.67	0.871>0.05 Not Significant
COMPLETION OF SENSORY BLOCK	10.10	1.26	10.53	0.89	0.132>0.05 Not Significant
ONSET OF MOTOR BLOCK	4.45	0.30	4.88	0.30	0.310>0.05 Not Significant
COMPLETION OF MOTOR BLOCK	29.58	1.04	31.45	0.91	0.128>0.05 Not significant

GRAPH 7: BLOCK CHARACTERISTICS

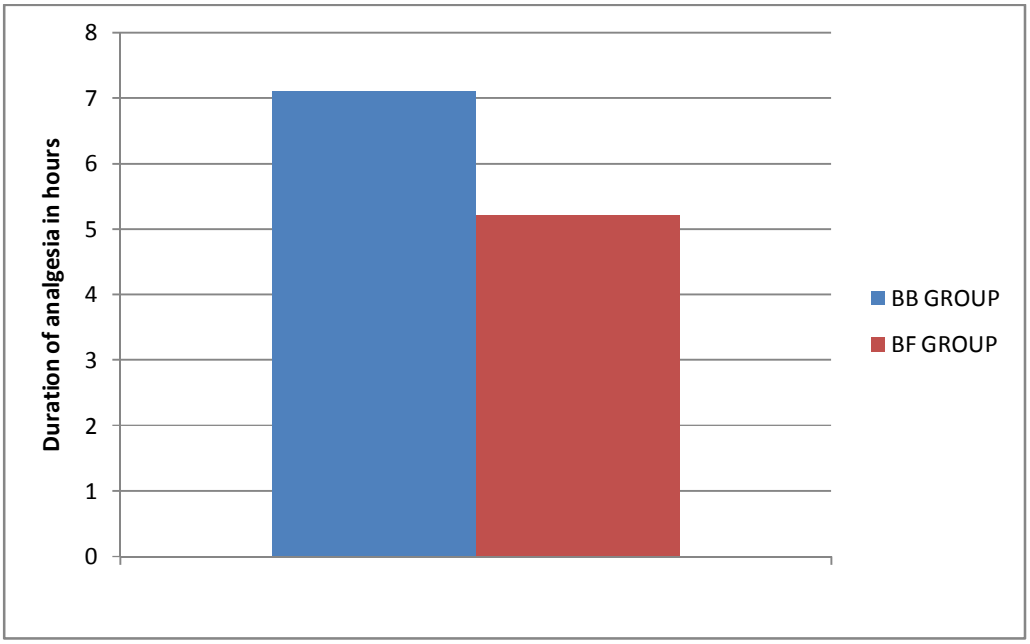


There is no significant statistical difference in the time of onset of sensory and motor block, completion of sensory and motor block in between group BB and group BF. Mean onset time of sensory block, completion of sensory block,onset of motor block and completion of motor block in group BB is 5.73minutes,10.1 minutes,4.45 minutes and 29.58 minutes respectively. In BF group, mean time of onset of sensory, completion of sensory block,onset of motor block and completion of motor block are 5.96mins,10.53mins,4.88mins and 31.45 mins respectively.

TABLE 11: COMPARISON OF DURATION OF ANALGESIA

PARAMETER	GROUP BB		GROUP BF		STATISTICAL INFERENCE
	MEAN HOURS	±S.D.	MEAN HOURS	±S.D	
DURATION OF ANALGESIA	7.1	1.008	5.2	1.080	0.001<0.05 SIGNIFICANT

GRAPH 8: DURATION OF ANALGESIA

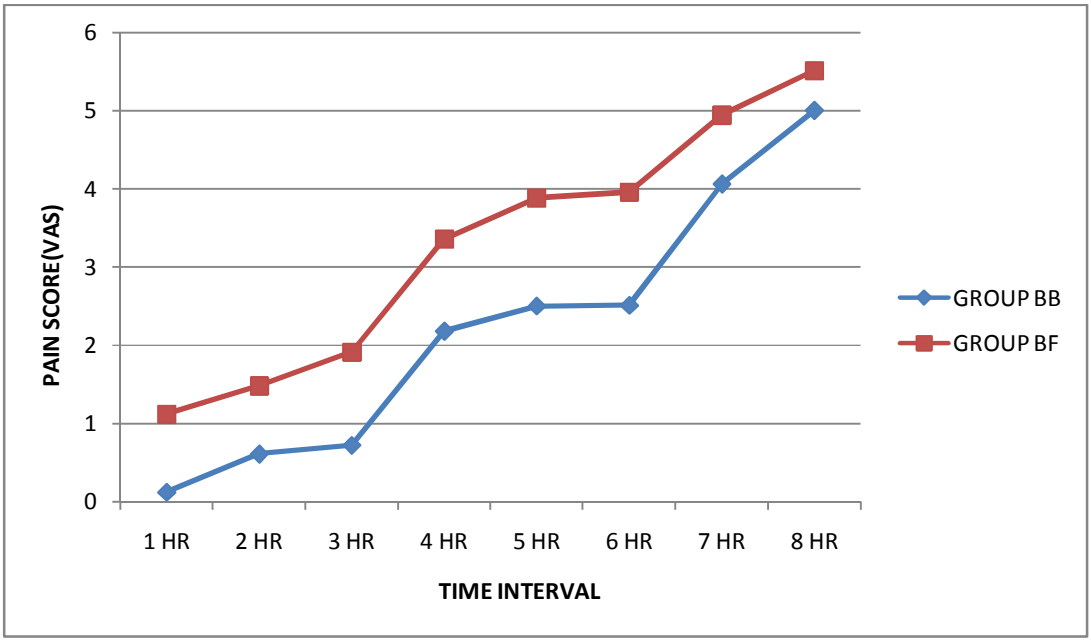


There is statistically significant difference in the duration of analgesia between group BB and group BF. The duration of analgesia was longest with butorphanol group(5-9 hours; mean-7.1 hours), whereas in group BF was 3-9 hours, mean 5.2 hrs.

TABLE 12: THE MEAN POST-OPERATIVE PAIN SCORES(VAS) AT DIFFERENT TIME INTERVALS IN GROUP BB & GROUP BF

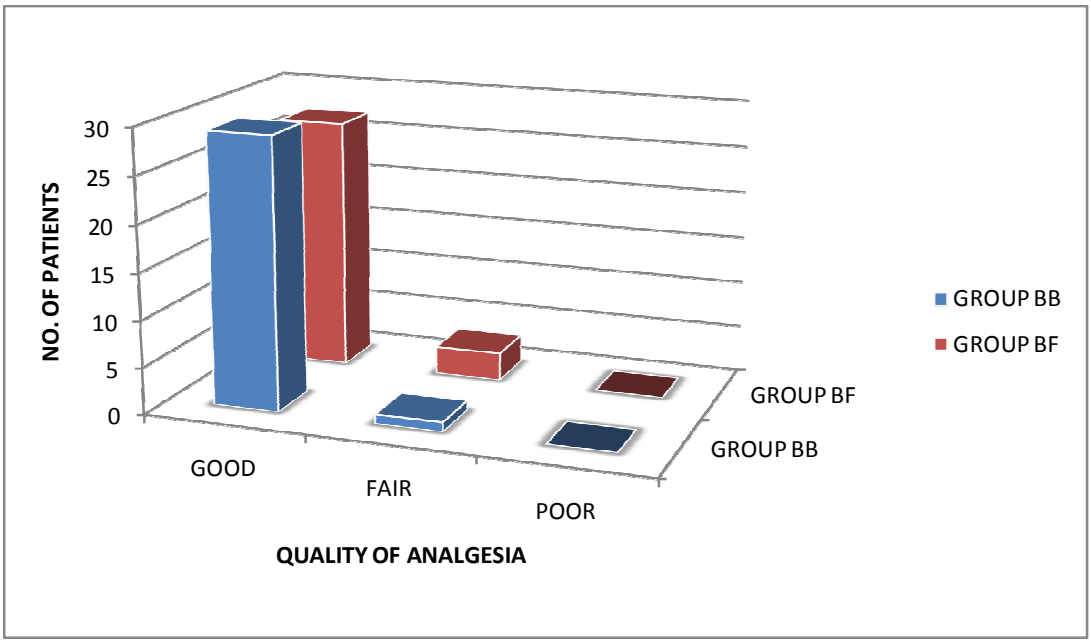
TIME INTERVAL	GROUP BB		GROUP BF		STATISTICAL SIGNIFICANCE
	MEAN	±S.D.	MEAN	±S.D.	
1 HR	0.12	0.71	1.12	0.45	0.000<0.05 S
2 HR	0.61	0.61	1.48	0.91	0.008<0.05 S
3 HR	0.72	0.60	1.91	0.56	0.021<0.05 S
4 HR	2.18	0.87	3.06	1.70	0.001<0.05 S
5 HR	2.50	1.17	3.88	0.96	0.003<0.05 S
6 HR	2.51	1.07	3.96	0.91	0.012<0.05 S
7 HR	4.06	0.61	4.94	0.56	0.001<0.05 S
8 HR	5.00	0.08	5.51	0.54	0.44>0.05 NS

GRAPH 9: PAIN SCORES (VAS) IN GROUP BB & BF



As shown in graph 9, the pain scores as assessed on VAS were low and remained low for a significant time in the post operative period in group BB when compared to group BF.

GRAPH 10: QUALITY OF ANALGESIA



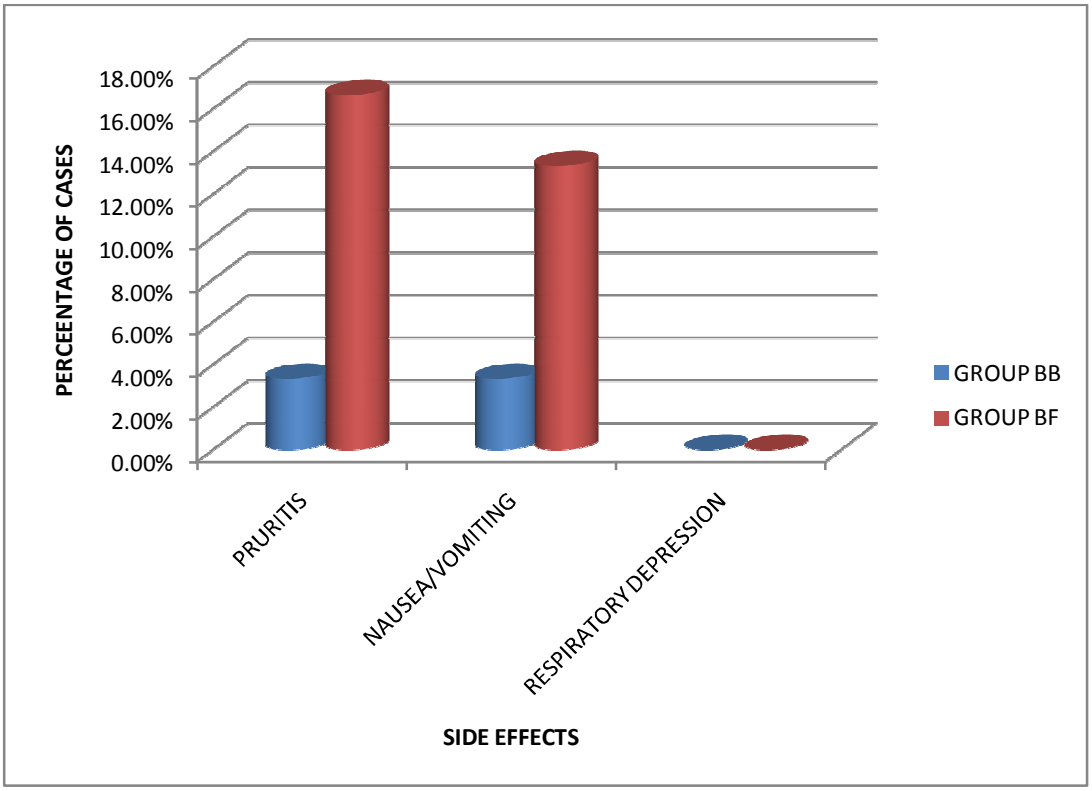
Majority of the patients in both group BB and Group BF had good quality of analgesia. None of the patients required top up doses of local anaesthetics intra operatively.

TABLE 13:

COMPARISON OF SIDE EFFECTS BETWEEN GROUPS BB & BF:

SIDE EFFECTS	GROUP BB (n=30)	GROUP BF (n=30)	p Value
Pruritis	1	6	0.353>0.05 NS
Nausea/vomiting	1	4	0.671>0.05 NS
Respiratory depression	0	0	0

GRAPH 11: COMPARISON OF SIDE EFFECTS:



In group BB, 3.33% of patients had nausea/vomiting compared to 13.33% of patients in BF group, whereas 3.33% of patients had pruritis in BB compared to 16.66% of patients in BF group. This was not statistically significant ($p > 0.05$). None of the patients in both the groups were reported to have respiratory depression.

DISCUSSION

Opioids are being extensively used as adjuvants to local anaesthetics to improve the quality of the block and to produce dose-sparing effect. Epidural administration of various analgesics have gained popularity following the discovery of opioid receptors in the spinal cord. The use of epidural techniques also offer the advantage of post-operative analgesia. There are a number of studies to prove the efficacy of adding opioids to local anaesthetics. Opioid receptors are found to be highly specific receptors located in specific regions of central nervous system and peripheral nervous system. The opioid receptors located in the dorsal horn of spinal cord mediate both pre and post synaptic effects modulating the nociceptive input without sensory or motor blockade. Epidural administration of opioids have found to be superior than intravenous or intramuscular injection of opioids.

Although in recent times various opioids have been used for post operative analgesia, earlier morphine and pethidine were the standard drugs, which were associated with increased incidence of delayed respiratory depression and abuse potential.Stimulation of spinal opiate receptors produces spinal analgesia with lesser side effects.

Butorphanol a mixed opioid , with an agonist and antagonist action at μ receptor and an agonist action at kappa receptor, is found to produce potent analgesia with fewer side effects and very low abuse potential. It is highly lipid soluble and has greater affinity to opioid receptors, which contributes to its greater potency and efficacy.

Fentanyl, being a synthetic opioid receptor agonist, is found to produce analgesia by binding to supra- spinal opioid receptors when administered into the epidural space. It is better retained in the epidural space because of its high lipid solubility. Following administration into the epidural space, systemic absorption occurs, but it has a shorter half life, hence there is less circulating plasma drug concentration. Epidural administration of fentanyl is associated with reduced respiratory depression and lesser incidence of side effects like nausea, vomiting and pruritis.

The present study is prospective ,randomized, comparative study done to compare the efficacy of butorphanol and fentanyl added as adjuvants to bupivacaine in epidural anaesthesia in lower abdominal surgeries with respect to intra operative hemodynamic stability and post operative analgesia. A total of 60 patients both male and female belonging to the age group of 20-60 years were studied, among which majority of the

patients were aged between 40-50 years and majority of the 60 patients underwent general surgery.

During the preoperative assessment, the patients were clearly explained about the anaesthetic procedure and also educated about the assessment of pain in the post operative period using VAS. Written consent was obtained from all the patients. At 10 pm on the night before surgery, all patients were premedicated with Tab. Alprazolam 0.25 mg and Tab. Ranitidine 150 mg and advised nil per oral from then onwards.Patients were randomly divided into two groups of 30 per group, Group BB- bupivacaine with butorphanol and Group BF- bupivacaine with fentanyl. All the surgeries were done under lumbar epidural anaesthesia with a total volume of 20 ml of study drug in each group. Intra operatively vital parameters like pulse rate, mean arterial blood pressure, oxygen saturation, respiratory rate were recorded every 5 minutes from the time of injecting the study drug upto 30 minutes and then onwards every 10 minutes upto 120 minutes. Similarly the sedation score throughout the intra-operative period, the time of onset and completion of sensory block, the time of onset and completion of motor block,side effects if any were recorded

There was no case of epidural failure and no patient required epidural top-up with local anaesthetics in the intra operative period. In the post- operative period pain was assessed using the VAS, every hour upto 8hrs.

At the same time the vital parameters and side effects were also recorded and treated accordingly. When the VAS > 5, the patients were given rescue analgesia with Tramadol 100mg in 10ml of normal saline through the epidural catheter and study in that patient ceased and the duration of analgesia was noted. The duration of analgesia was significantly seen to be prolonged with bupivacaine- butorphanol group (BB)

IN OUR STUDY:

INTRA OP HEMODYNAMICS:

In our study,the majority of patients were hemodynamically stable intra-operatively. Comparison of heart rate and MAP within the groups was done using paired ‘t’ test whereas comparison of heart rate and MAP in between the two groups was done using unpaired ‘t’ test.

The mean arterial BP in Group BB was 83.4 mm Hg± 1.26 (S.D) mmHg and in group BF was 81.3mm Hg± 1.05 (S.D) mm Hg. The mean reduction in MAP was statistically insignificant between both the groups. In both groups BB and BF, there was a fall from the baseline MAP of 92.7 mmHg±6.08(S.D.) and 89.7 mmHg±7.27(S.D.) respectively to 78.8mmHg and 76.7mmHg at 40-50 mins time interval which was not statistically significant.

The mean pulse rate in Group BB was 75.6±1.35(S.D.)/min and in Group BF was 74.67±0.92(S.D.)/min. The statistical analysis showed that there was no significant difference between the two groups.

The mean respiratory rate in group BB was 12.6± 1.32(S.D.)/min and in group BF was 12.9±0.98(S.D.)/min. The statistical study showed no significant difference in the mean respiratory rate between the 2 groups.

Oxygen saturation (SPO_2) maintained between 98-97% in both the groups. None of the patients in both the groups showed desaturation(SPO_2<95%).

Our study can be compared to the following studies:

Gough et al., in 1988 used epidural fentanyl 1.5µg/ kg body weight in 10ml of sterile solution and concluded that the range of mean(S.D) of cardio- respiratory variables like heart rate 84(±2) to 95(±18) beats/ min, systolic BP of 121(±19) to 133(±14) mm of Hg, diastolic BP of 70(±10) to 76(±10) mm of Hg and RR of 14(±3) to 16(±4) / min varied negligibly from basal recordings.

Premila Malik, Chhavi Manchanda, Naveen Malhotra et al., in 2006 conducted a study to assess and compare the safety and efficacy of postoperative analgesia with epidural butorphanol 2mg and fentanyl 50µg. Their study showed that there was no significant changes in pulse rate, systolic and diastolic BP, RR and SpO2 in the 2 groups at different time intervals throughout the 24 hours study period ($p > 0.05$).

SEDATION SCORES:

Catherine O Hunt in her study has reported a higher incidence of sedation with epidural butorphanol and is a dose dependent side effect. In our study sedation scores were higher with butorphanol group as compared with fentanyl group.Mean value of subjective sedation score was 1.00±0.06 in group BF and 3.0±0.64 in group BB. Majority of the patients

had mild sedation. The patients were awake but drowsy. This was statistically significant ($p < 0.001$).

JS Naulty in his study noted that sedation was significant, but was of mild type (arousable with verbal response).72% of patients on epidural butorphanol 2mg had clinically significant sedation in a study by **Therese K et al.**

Rutter DV et al., in 1981 reported that fentanyl 100µg for postoperative pain relief produced increase in sedation.

ONSET AND COMPLETION OF SENSORY BLOCK:

In our study the onset and completion of analgesia was hastened with the addition of butorphanol and fentanyl. But there was no statistically significant difference detween the two groups. Addition of 1mg butorphanol to 20ml 0.5% bupivacaine reduced the latency of onset of analgesia to 5-9mins and completion of analgesia occurred earlier(9-14 mins, mean 10.10 mins). In BF group also the onset of analgesia was rapid(5-10mins; mean 5.95 mins) and completion of analgesia occured in (9-15mins;mean 10.96 mins).

Mok et al., in 1986 did a study to evaluate the analgesic efficacy and safety of epidural butorphanol 4mg in comparison to that of epidural morphine 5mg in patients with postoperative pain. Onset of pain relief with epidural butorphanol appeared at 15 minutes and peaked at 30 minutes.

Maurice Lippmann in 1988 has reported in his study that epidural butorphanol 4mg used for postoperative analgesia in non- obstetric abdominal surgeries has produced analgesia within 15 minutes.

Abboud et al. in 1986 studied the efficacy of epidural butorphanol for post operative pain relief and reported the onset of analgesia with 1mg butorphanol was 15mins.

Cousins and Mather et al. in 1984 reported the time of onset of analgesia with epidural fentanyl 100µg to be 5-10mins.

Rutter DV et al., in 1981 reported that 100µg of epidural fentanyl for postoperative pain relief had a rapid onset of action i.e almost 50% reduction in mean pain within 5 minutes.

In a study by **Lomessay A et al.,** in 1984 concluded that epidural fentanyl 200µg provides rapid analgesia that remains optimum during 2 hours despite the intensity and pain stimulation.

Naulty JS et al., in 1985 used different doses of epidural fentanyl in parturients following caesarean delivery. They concluded that fentanyl 100µg produced pain scores of 0 in 3-6 minutes.

QUALITY OF ANALGESIA:

In our study majority of the patients in both group BB and group BF reported good quality of analgesia. None of the patients in both the groups required top up dose of local anaesthetics intra operatively.

Quisqueya T et al., in 1991 compared epidural butorphanol in doses of 1mg, 2mg and 4mg with morphine 5mg. He concluded that each dose of butorphanol produced greater pain relief than morphine at 15, 30, 45 and 60 minutes($p < 0.05$).

Lytle SA et al., in 1991 did a retrospective analysis with fentanyl (50µg) and showed that epidural fentanyl provides good to excellent pain relief.

Sugimoto M et al., in 1997 compared the degree of analgesia using different doses of epidural fentanyl and found that epidural fentanyl 25µg provided superior analgesia than 12.5µg.

Hwang KB, Chung CJ, Lee et al., in 2004 compared analgesic efficacy of epidural butorphanol and epidural fentanyl and concluded that there was no significant difference in the quality of analgesia between the two groups.

POST OPERATIVE PAIN SCORES:

In our study the pain scores as assessed on the VAS were low and remained low for a significant time in the post operative period in both group BB and group BF. The range of post operative pain scores in group BB at 1, 2,3,4,5,6,7 hours were between 0-5, where as in BF group for the same time interval was between 3-6. There was a statistically significant difference in pain score in between both the groups.

DURATION OF ANALGESIA:

In our study the duration of analgesia was significantly prolonged with the addition of opioids to local anaesthetics. The mean duration of analgesia with the group BF was 5.2 hours, where as in Group BB was 7.1 hours. Our study was consistent with those observed by **Cousins and Marther et al**.,1984 and **Peach et al.,** in 1990, who observed the mean duration of analgesia with epidural fentanyl was 5.7 hours and 5.2 hours respectively. **Malik et al .,** in 2006 studied the duration of analgesia with

epidural butorphanol with varying doses and observed that epidural butorphanol produced a significantly longer duration of analgesia when compared to fentanyl.

SIDE EFFECTS:

Narcotics are well known for their potential side effects such as pruritis, nausea, vomiting, urinary retention and respiratory depression. Delayed respiratory depression is one of the most troublesome of these side effects.

Pruritis: In our study 3.33% of patients in butorphanol group had pruritis and whereas 16.66% of patients in fentanyl group had pruritis which was statistically insignificant ($p>0.05$).

In a study by **Ackermann et al.,** in 1989, 7% of patients reported pruritis with 2mg of epidural butorphanol and in a study by Palacios et al in 1991, 1.4% of patients reported pruritis with 2mg of butorphanol.

In a study by **Lytle SA et al.,** in 1991 using fentanyl 50µg ,4% of patients had pruritis.

Nausea and vomiting:

In our study 3.3% of patients in butorphanol group had nausea whereas in fentanyl group 13.33% of patients had nausea which was insignificant statistically ($p>0.05$).

No patients on epidural butorphanol had nausea or vomiting in separate studies conducted by **JS Naulty et al., and Catherine O Hunt et al**. In a study by Lytle SA et al., in 1991, nausea was reported in 25.5% of cases.

Premila Malik, Chhavi Manchanda, Naveen Malhotra in 2006 compared the efficacy of epidural butorphanol 2mg and fentanyl 50µg found that the incidence of nausea and vomiting was higher in fentanyl group.

Respiratory depression: In our current study, none of the patients in butorphanol group or fentanyl group reported respiratory depression which was consistent with the following studies.

No patients had respiratory depression with butorphanol in studies conducted by **Maurice Lippmann et al**., in 1988, **Catherine O Hunt et al in 1989, JS Naulty et al., in 1989.**

Rutter DV et al., in 1981 reported decrease in respiratory rate in patients who received 100µg of fentanyl.

Negre I et al., in 1987 observed the effect of 200µg of fentanyl on ventilatory response to carbon dioxide and concluded that fentanyl induces a non systemic ventilatory response that may be due to rostral spread of the drug.

SUMMARY

This prospective randomized controlled clinical comparative study entitled "**A COMPARATIVE STUDY OF EPIDURAL BUTORPHANOL AND EPIDURAL FENTANYL AS ADJUVANTS TO BUPIVACAINE IN LOWER ABDOMINAL SURGERIES "** was conducted in 60 patients of either sex, aged between 20- 60 years of ASA grade I and II admitted for elective surgeries to Thanjavur Medical College, from June 2012 to July 2014.

Written informed consent was taken and pre-anaesthetic evaluation was done. All cases were given epidural anaesthesia using 0.5% bupivacaine with butorphanol 1mg(total volume of 20 ml) or 0.5% bupivacaine with fentanyl 100µg(total volume of 20ml) depending on study group BB or BF. In the perioperative period the following parameters were observed:

1. Vital parameters- heart rate, Spo_2, blood pressure and respiratory rate
2. Onset and completion of analgesia
3. Quality of analgesia
4. Duration of analgesia
5. Sedaton score

6. Side effects

In the postoperative period, intensity of pain was assessed using Linear Visual analog scale.

Demographic profile (age, sex) was comparable in both groups.

INTRA OPERATIVE HEMODYNAMICS:

The mean arterial blood pressure in group BB was 83.4 mm Hg ± 1.26 (S.D) and in group BF was 81.3mmHg±1.05(S.D).

The mean pulse rate in Group BB was 75.6±1.35(S.D.)/minutes and in Group BF was 74.67±0.92(S.D.)/minutes

The mean respiratory rate in group BB was 12.6± 1.32(S.D.)/min and in group BF was 12.9±0.98(S.D.)/min. The statistical study showed no significant difference in the mean arterial blood pressure,mean pulse rate, mean respiratory rate between the 2 groups.

Oxygen saturation (SPO_2) maintained between 97-98% in both the groups. None of the patients in both the groups showed desaturation(SPO_2<95%).

Onset and completion of analgesia:

In BB group mean onset of analgesia was 5-9mins and completion of analgesia occurred earlier(9-14 mins, mean 10.10 mins). In BF group also the onset of analgesia was rapid(5-10mins; mean 5.95 mins) and completion of analgesia occured in (9-15mins;mean 10.96 mins). But there was no statistically significant difference between the two groups.

Duration of analgesia: The duration of analgesia was longer in butorphanol group which ranged from 5to8 hours with a mean of 7.6 hours compared to fentanyl group which ranged from 3 to 7 hours with a mean of 5.8 hours.This was clinically and statistically significant ($p< 0.001$).

Quality of analgesia:The quality of analgesia was good in both BB and BF group.There was no statistical significance between both the groups.

Sedation score:

The mean value of subjective sedation score was 1.00±0.06 in group BF and 3.0±0.64 in group BB. This was statistically significant ($p< 0.001$).

Side effects

The frequency of pruritis, nausea and vomiting was more in fentanyl group when compared to butorphanol group but this was not statistically significant. Respiratory depression was not reported in both the groups.

CONCLUSION

It can be concluded from the above study that epidural butorphanol provides a longer duration of good quality of analgesia with fewer side effects like sedation which are statistically significant when compared to epidural fentanyl.

In view of its safety profile, epidural butorphanol can be routinely employed as an adjuvant to bupivacaine in epidural anaesthesia for good intraoperative and postoperative analgesia for various surgical procedures. However more studies with different dosages and different techniques (epidural bolus and infusion) of both the study drugs should be conducted to evaluate the efficiency and to conclude the above facts.

BIBILIOGRAPHY

1. Dr. Nirvil Pal,Dr.Kiran malhotra.Dr.Chitra et al, effect of morphine on post op respiratory function. Comparison between systemic and epidural routes,Indian J. Anaesthesia 2004;48(3):204-206
2. Admir Hadzic-Textbook of regional anaesthesi and acute pain management edition 4,230-264.
3. Hunt CO,Naulty,Mallinow AM,Datta S, Ostheimer GW. Epidural butorphanol-bupivacaine for analgesia during lobor and delivery. Anesth Analg 1989.
4. Stein C:The control of pain in peripheral tissues by opioids. N England J Med 1995;332:1685
5. Ronald D Miller, Miller's a=Anaesthesia, 7th edition, vol 2:2422-2426
6. Anatomy for anaesyhesiologists-Harold Ellis.8th edition.95-125
7. Bromage P: epidural anaesthesia.WB Saunders,1978
8. Cousins M.Veering b: Epidural Neural Blockade. In Cousins M, Bridenbaugh P(eds): Neural Blockade.3rd ed.Lippincott-Raven.1998;243-320
9. Meltem Yilmaz nad Cynthia A. Wong-Spinal And Epidural anaesthesia, Edition 4, 35-52

10. Cousins, Michel J, Bernadette T. Veering Epidural Neural Blockade: Neural Blockade In Clinical Anaesthesia and Management of Pain, Philadelphia: Lippincott Raven 1998; 243-321.
11. Leon Visser. Epidural Anaesthesia. Anaesthesia update 2001; 13(11): 1-4.
12. Micheal J cousins, Bernadette T. Veering. Epidural neural blockade Chapter 8.Neural blockade in Clinical Anaesthesia and management of pain, third edition 1998; 243-312.
13. David L Brown. Spinal, Epidural and Caudal Anaesthesia, chapter 42.
14. Anaesthesia Ronald D.Miller 5th edition 1491-98.
15. Bonika, John J. Definition and Taxonomy of pain. Chapter-2 : Management of pain, 2nd Edn, Philadelphia : Lea & Febiger 1990; 18-27.
16. Ronald Melzack – Text book of pain, 4th edition.
17. Chapman C Richard, Karen L, Syrjala. Measurement of Pain - The Management of Pain. John J, Bonica. 2nd Edn, Philadelphia. Lea and Fabiger 1990: 580-94.
18. Dolin Sj,Cashman JN,Bland JM: Effectiveness of Acute Postoperative Pain Management:I Evidence from published data. Br j Aneasth 2002;29:409
19. Liu S, Carpenter RL, Neal JM: Epidural Anaesthesia and Analgesia. Their role in postoperative outcome. Anaesthesiology 1995;82:1474

20. Peter L Bailey, Talmage D Egan, Theodore H Stanley. Intravenous opioid Anaesthetics chapter 10, Ronald D. Miller Anaesthesia 5th edition, vol 1, Churchill Livingstone 2000; 344-46.

21.Howard B Gutstan, Huda Akil. Opioid Analgesics, 10th edition. Goodman and Gillman's The pharmacological basis of therapeutics. 2001; 590-601.

22.Robert K Stoelting. Opioid agonists and antagonists, Pharmacology and physiology in anesthetic practice, 3rd edition. Lippincott Raven 1999; 103 –05.

23.Pert CB, Synder SH. Opiate receptor: demonstration in nervous tissue. Science 1973 ; 179: 1011-4.

24.Cousins MJ, Mather Le. Intrathecal and epidural administration of opioids. Anaesthesiology 1984; 61: 276-310.

25.Howard B. Gutstein, Huda Akil. Opioid analgesics. Goodman & Gillman's The pharmacological basis of therapeutics.Goodman Gilman Alfred, Hardman G. Joel, Lee E. Limbrid. 10th Edn. USA: McGraw Hill; 2001, 569-629pp.

26,Stoelting Robert K. Opioid agonists and antagonists. Pharmacology and physiology in anesthetic practice. 3rd Edn. USA: Lippincott – Raven; 1999, 77-79 pp.

27.Kazuhiko Fukuda. Intravenous opioid anesthetics. Anesthesia. Ronald D. Miller. 6th edn. USA: Churchill Livingstone; 2005, 379-425 pp

28.Robert K Stoelting. Pharmacology and Physiology in anaesthesia practice, 3rd edition. Lippincott – Raven publishers (United States of America) 1999; 628-33.

29.Naulty J, Stephen, Sanjay Datta, Ostheimer Gerard W, Mark D, Jhonson and Gerald A Burger. Epidural fentanyl for postcaeserean delivery pain management. Anaesthesiology 1985; 63(6):694-8.

30. Katz J, Melzak R. Measurement of pain. Surg Cli Nor 1999; 79:231-52.

31. Behar M, Magora F, Olshwang D, Davidson JT. Epidural morphine in treatment of pain. Lancet 1979 ; 1: 527-28.

32. Ameer B, Slater FJ. Drug therapy reviews: evaluation of butorphanol tartrate. Am J Hosp Pharm Dec. 1979; 36(12) : 1683-91.

33.JS Naulty, S Weintraub, J Mcmahon, Ostheimer C Hunt, R Chantigian. Epidural butorphanol for postcesarean delivery pain management. Anaesthesiology 1984; Sept: A 415.

34.Naulty J, Stephen, Sanjay Datta, Ostheimer Gerard W, Mark D, Jhonson and Gerald A Burger. Epidural fentanyl for postcaeserean delivery pain management. Anaesthesiology 1985; 63(6):694-8.

35. Mok MS , Tsai YJ, et al. Efficacy of epidural butorphanol compare to morphine for the relief of postoperative pain. Anaesthesiology 1986; 65: A 175.

36.Ackerman WE, Junje MM, Colclough GW. A comparison of epidural fentanyl, buprenorphine and butorphanol for the management of postcesarean section pain. Anaesthesiology 1988; sept; 34: A 401.

37.Maurice Lippmann, Martin S Mok. Epidural butorphanol for relief of postoperative pain. Anaesth Analg 1998; 67:418-21

38.Catherine O Hunt, J Stephen Naulty, Andrew M Malinow, Sanjay datta, Gerard W Ostheimer. Epidural butorphanol-Bupivacaine for analgesia during labour and delivery. Anaesth Analg 1989; 68:323-7.

39. Salomaki, Timo E, Jorma D Laitinen, Lauri S. Nutinen. A radomized double blind comparison of epidural versus intravenous fentanyl infusion for analfesia after thoracotomy. Anaethesiology 1991; 75(5): 790-5.

40. Lytle SA, Goldsmith DM, Neuendorf TL, Lowry ME. Postoperative analgesia with epidural fentanyl. J Am Osteopath Assoc 1991 ; 91(6):547-50.

41. Quisqueya T, Palacios, Monica M Jones, Joy L, Hawkins, Jayshree N., Adenwala, Stephen Longmire, Kenneth R Hess, Barbara S. Sknjonsby et al., Postcesarean section analgesia: a comparison of epidural butorphanol and morphine. Can J Anaesth 1991: 38(1): 24-30.

42. Palacios, Monica M Jones, Joy L Hawkins, Jayshree N Adenwala, Stephen Longmire, Kenneth R Hess, Barbara S Sknjonsby et al., Post cesarean section analgesia: a comparison of epidural butorphanol and morphine. Can J Anaesth 1991; 38(2): 43-47.

43. Hwang KB, Chung CJ, Lee JH, Lee SC, Oh SH. Comparison of Butorphanol and Fentanyl for Patient-Controlled Epidural Analgesia after Gastrectomy. J Korean Pain Soc 2004 Jun; 17(1): 24-48.

44. Premila Malik, Chhavi Manchanda, Naveen Malhotra. Comparative Evaluation of Epidural Fentanyl and Butorphanol for Postoperative Analgesia.J Anaesth Clin Pharmacol 2006; 22(4):377-382

45. Gough JD. The control of post-thoracotomy pain- A comparative evaluation of thoracic epidural fentanyl infusions and cryoanalgesia. Anaesthesia 1988 ; 43: 780-83

PROFORMA

NAME: WEIGHT:

AGE/SEX: DIAGNOSIS:

IP.NO: TYPE OF SURGERY:

DATE: ASA GRADE:

GROUP:

PRE-OPERATIVE EVALUATION:

PULSE RATE: AIRWAY:

BLOOD PRESSURE: CVS:

RR: RS:

PRE-OPERATIVE INVESTIGATIONS:

Hb%:

BT/CT:

BLOOB SUGAR:

BLOOB UREA:

SERUM CREATININE:

ECG:

X-RAY CHEST:

PRE MEDICATION:

ANAESTHETIC TECHNIQUE:

DRUG DOSE:

DURATION OF SURGERY:

TOP UP DOSE(IF ANY):

PARAMATERS STUDIES:

1.ONSET OF SENSORY BLOCK: 4.QUALITY OF ANALGESIA

2.COMPLETION OF SENSORY BLOCK: 5.DURATION OF ANALGESIA:

3.LEVEL OF SENSORY BLOCK: 6, SIDE EFFECTS.

VITAL PARAMETERS: INTRA OPERATIVE

TIME mins	**0**	**5**	**10**	**15**	**20**	**25**	**30**	**40**	**50**	**60**	**70**	**80**	**90**	**100**	**110**	**120**
HR																
SBP/DBP																
MAP																
SPO_2																
RR																
SEDATION SCORE																
SIDE EFFECTS																

POST OPERATIVE PERIOD:

TIME (HOURS)	**1st**	**2nd**	**3rd**	**4th**	**5th**	**6th**	**7th**	**8th**
HEART RATE								
SBP/DBP								
MAP								
RR								
SPO_2								
SEDATION SCORE								
PAIN SCORE								
SIDE EFFECTS								

DURATION OF ANALGESIA:

KEY TO MASTER CHART

M	MALE
F	FEMALE
PR	PULSE RATE
MAP	MEAN ARTERIAL PRESSURE
SPO_2	OXYGEN SATURATION
RR	RESPIRATORY RATE
SS	SEDATION SCORE
VAS	VISUAL ANALOG SCALE
GROUP BB	BUPIVACAINE-BUTORPHANOL
GROUP BF	BUPIVACAINE- FENTANYL
SB	SENSORY BLOCK
MB	MOTOR BLOCK
Y	YES
N	NO
MINS	MINUTES
HRS	HOURS

SL.NO	NAME	AGE	SEX	WEIGHT(Kgs)	TYPE OF SURGERY	DURATION OF SURGERY IN MINS	GROUP	PULSE RATE 0MINS	PULSE RATE 5MINS	PULSE RATE 10MINS	PULSE RATE 15MINS	PULSE RATE 20MINS	PULSE RATE 25MINS	PULSE RATE 30MINS	PULSE RATE 40MINS	PULSE RATE 50MINS	PULSE RATE 60MINS	PULSE RATE 70MINS	PULSE RATE 80MINS	PULSE RATE 90MINS	PULSE RATE 100MINS	PULSE RATE 110MINS	PULSE RATE 120MINS	MAP 0MINS	MAP 5MINS	MAP 10MINS	MAP 15MINS
1	RAMASAMY	40	M	45	GENERAL SURGERY	85	BB	88	85	80	78	76	76	74	70	70	70	72	68	70	72	72	72	90	92	82	73
2	MANI	40	M	50	GENERAL SURGERY	80	BB	85	85	80	77	74	72	72	70	68	64	64	62	66	70	74	74	94	87	77	76
3	MAHALINGAM	45	M	50	GENERAL SURGERY	80	BB	95	84	76	76	72	70	70	68	68	64	64	62	62	66	62	68	99	98	92	90
4	RAJAMANI	40	F	48	GENERAL SURGERY	90	BB	92	88	79	78	72	74	70	70	66	64	62	62	60	60	68	70	102	101	98	95
5	DHARMARAJ	45	M	45	GENERAL SURGERY	90	BB	74	76	72	70	68	68	66	64	64	64	62	62	60	64	70	68	82	80	84	78
6	RAJASEKAR	38	M	36	GENERAL SURGERY	95	BB	102	98	96	90	80	82	85	89	76	72	74	76	88	86	89	86	96	90	91	88
7	KUMARESAN	35	M	38	GENERAL SURGERY	85	BB	84	80	75	72	70	69	70	72	72	74	74	72	76	70	72	78	89	89	87	85
8	SHANKAR	42	M	52	GENERAL SURGERY	80	BB	92	90	90	88	86	84	82	80	80	78	78	76	76	74	72	74	77	75	72	70
9	SIVAM	45	M	55	GENERAL SURGERY	75	BB	98	97	96	94	92	90	88	86	84	82	82	82	80	78	76	74	94	87	86	84
10	BALASUBRAMANIAN	50	M	45	GENERAL SURGERY	80	BB	78	76	76	74	72	70	68	68	66	69	69	69	64	66	64	62	96	102	92	90
11	SIVASANKAR	38	M	48	GENERAL SURGERY	95	BB	86	84	82	80	78	78	76	74	72	70	68	68	68	66	64	62	94	92	90	90
12	GANAPATHY	35	M	43	GENERAL SURGERY	80	BB	84	82	80	78	78	76	72	70	66	68	68	64	64	62	66	64	98	98	98	94
13	PARVATHI	42	M	50	GENERAL SURGERY	85	BB	99	96	94	92	90	88	86	84	82	82	82	84	80	78	76	74	102	100	96	96
14	AROKIARAJ	45	F	52	GENERAL SURGERY	85	BB	104	100	98	95	92	90	89	86	88	86	84	84	82	80	78	76	92	92	88	86
15	VEDHAMOORTHI	40	M	38	GENERAL SURGERY	80	BB	100	98	96	94	92	90	88	86	84	82	82	82	80	78	76	74	90	90	84	82
16	JESUDAS	34	M	35	GENERAL SURGERY	70	BB	78	76	76	78	74	72	70	70	68	68	68	66	64	62	60	66	94	92	88	88
17	MURUGAIYAN	35	M	48	GENERAL SURGERY	75	BB	86	84	82	80	78	76	74	74	74	76	74	78	76	76	72	72	100	102	96	91
18	RANGANATHAN	42	M	55	GENERAL SURGERY	75	BB	98	96	94	92	90	90	88	86	86	84	84	84	82	80	78	76	84	80	76	73
19	RATHINAM	45	M	50	GENERAL SURGERY	85	BB	92	90	88	86	85	88	84	80	78	76	76	74	72	70	72	70	92	90	91	89
20	BABU	48	F	50	GENERAL SURGERY	90	BB	88	84	80	78	78	80	84	86	86	84	84	84	82	80	78	76	88	86	80	78

SL.NO	NAME	AGE	SEX	WEIGHT(Kgs)	TYPE OF SURGERY	DURATION OF SURGERY IN MINS	GROUP	PULSE RATE 0MINS	PULSE RATE 5MINS	PULSE RATE 10MINS	PULSE RATE 15MINS	PULSE RATE 20MINS	PULSE RATE 25MINS	PULSE RATE 30MINS	PULSE RATE 40MINS	PULSE RATE 50MINS	PULSE RATE 60MINS	PULSE RATE 7OMINS	PULSE RATE 80MINS	PULSE RATE 90MINS	PULSE RATE 100MINS	PULSE RATE 110MINS	PULSE RATE 120MINS	MAP 0MINS	MAP 5MINS	MAP 10MINS	MAP 15MINS
21	KUMARVEL	45	M	50	GENERAL SURGERY	95	BB	102	100	99	98	96	92	90	88	86	84	82	82	80	84	88	84	101	96	94	92
22	VELAUDHAM	42	M	57	GENERAL SURGERY	90	BB	112`	99	98	96	96	94	92	90	88	88	86	86	84	86	84	82	89	85	82	80
23	KARNAN	40	M	50	GENERAL SURGERY	90	BB	100	98	96	94	96	96	98	88	86	89	88	88	84	86	84	86	98	98	96	92
24	PAULRAJ	36	M	49	GENERAL SURGERY	85	BB	98	96	94	92	90	90	92	90	88	88	88	90	88	86	84	82	96	94	90	90
25	PETER SELVAM	38	M	49	GENERAL SURGERY	85	BB	90	88	86	86	84	84	82	80	80	78	76	76	74	72	70	70	98	98	96	94
26	SENAPATHY	46	M	48	GENERAL SURGERY	80	BB	74	72	70	68	66	66	64	62	60	60	62	62	62	64	64	62	94	94	90	90
27	GANDHIMADHY	48	M	50	GENERAL SURGERY	85	BB	72	70	69	68	68	66	66	64	64	62	62	60	62	64	60	60	93	88	86	84
28	PETHAN	45	M	55	GENERAL SURGERY	85	BB	78	76	76	70	68	68	66	66	64	67	67	68	62	62	60	62	90	88	88	84
29	VEERAPANDI	40	M	55	GENERAL SURGERY	90	BB	76	74	72	70	70	68	68	66	66	64	64	64	62	62	60	64	86	82	80	76
30	THIYAGARAJAN	45	M	50	GENERAL SURGERY	90	BB	98	98	96	94	94	92	92	90	88	86	84	84	82	80	80	78	85	80	78	78
31	KRISHNAMOORTHY	45	M	50	GENERAL SURGERY	80	BF	88	86	86	84	84	84	82	82	80	80	80	78	77	76	78	80	94	94	90	90
32	RAJANGAM	48	M	55	GENERAL SURGERY	85	BF	90	90	88	86	85	83	85	84	88	88	86	86	84	84	88	86	93	88	86	84
33	MARIMUTHU	36	M	46	GENERAL SURGERY	90	BF	94	92	92	90	88	86	86	84	88	90	88	88	86	86	88	90	90	88	88	84
34	SAIVARAJ	40	M	45	GENERAL SURGERY	90	BF	90	88	88	86	86	86	84	82	84	86	86	88	86	86	82	84	86	82	80	76
35	PANEERSELVAM	48	M	50	GENERAL SURGERY	85	BF	88	86	84	80	84	84	86	84	82	82	82	82	80	80	82	82	85	80	78	78
36	KENNADY	36	M	48	GENERAL SURGERY	90	BF	100	102	98	96	96	98	94	92	90	88	88	86	88	88	90	92	94	92	88	88
37	MAHALINGAM	38	M	45	GENERAL SURGERY	85	BF	106	102	100	100	96	94	92	88	86	88	88	88	90	90	92	90	100	102	96	91
38	KALAVATHY	42	F	45	GENERAL SURGERY	75	BF	90	88	89	86	88	89	88	88	86	86	84	84	84	82	82	84	84	80	76	73
39	SIVASANKAR	35	M	50	GENERAL SURGERY	75	BF	88	88	86	86	86	84	84	86	86	84	84	84	82	80	80	82	92	90	91	89
40	PERUMANANDHAM	32	M	55	GENERAL SURGERY	80	BF	72	72	70	70	68	66	64	62	66	66	66	66	64	62	62	64	88	86	80	78
41	GOVINDARAJ	40	M	49	GENERAL SURGERY	80	BF	78	78	76	74	72	70	70	70	68	66	64	64	66	66	64	68	101	96	94	92

SL.NO	NAME	AGE	SEX	WEIGHT(Kgs)	TYPE OF SURGERY	DURATION OF SURGERY IN MINS	GROUP	PULSE RATE 0MINS	PULSE RATE 5MINS	PULSE RATE 10MINS	PULSE RATE 15MINS	PULSE RATE 20MINS	PULSE RATE 25MINS	PULSE RATE 30MINS	PULSE RATE 40MINS	PULSE RATE 50MINS	PULSE RATE 60MINS	PULSE RATE 70MINS	PULSE RATE 80MINS	PULSE RATE 90MINS	PULSE RATE 100MINS	PULSE RATE 110MINS	PULSE RATE 120MINS	MAP 0MINS	MAP 5MINS	MAP 10MINS	MAP 15MINS
42	MADHIALAGAN	44	M	50	GENERAL SURGERY	85	BF	88	88	86	86	86	84	84	82	80	80	80	80	82	82	80	80	89	85	82	80
43	PERUMAL	49	M	50	GENERAL SURGERY	90	BF	100	98	96	96	94	92	92	88	90	90	90	92	92	90	90	90	98	98	96	92
44	JOHN	35	M	56	GENERAL SURGERY	95	BF	98	98	96	96	94	92	90	88	90	90	90	89	88	90	90	94	96	94	90	90
45	KATHIRAVAN	32	M	48	GENERAL SURGERY	60	BF	80	78	75	76	82	88	80	78	78	80	80	80	82	80	80	80	98	98	96	94
46	LEELAVATHY	46	F	45	GYNAECOLOGICAL	80	BF	88	86	84	82	80	86	88	84	82	80	80	80	78	78	77	80	90	92	82	73
47	VELMURUGAN	48	M	45	GENERAL SURGERY	85	BF	90	89	88	84	84	82	80	78	76	80	80	80	84	82	84	82	94	87	77	76
48	PANDIYAN	30	M	46	GENERAL SURGERY	90	BF	88	88	86	84	84	82	80	80	78	76	76	76	74	72	72	70	99	98	92	90
49	MOORTHY	39	M	45	GENERAL SURGERY	90	BF	100	89	98	96	94	94	94	92	90	90	90	90	88	88	86	86	102	101	98	95
50	MURUGESAN	44	M	42	GENERAL SURGERY	90	BF	88	88	86	84	82	82	80	80	78	78	78	78	76	74	78	80	82	80	84	78
51	RAJAMANI	49	M	38	GYNAECOLOGICAL	95	BF	114	112	98	94	90	90	88	90	88	88	86	86	84	86	84	82	90	88	86	86
52	PETHAIYAN	40	M	40	GENERAL SURGERY	90	BF	100	98	96	94	92	92	90	88	86	86	86	86	84	82	86	86	78	78	74	72
53	THANGARAJ	45	M	45	GENERAL SURGERY	85	BF	78	76	72	70	68	70	70	72	72	70	72	72	72	70	72	70	96	90	91	88
54	VEERAMANI	50	M	42	GENERAL SURGERY	80	BF	76	76	74	78	78	76	76	74	72	72	72	70	70	72	72	72	89	89	87	85
55	KANNAIYAN	52	M	55	GENERAL SURGERY	75	BF	88	86	84	84	82	82	80	80	78	76	76	76	74	76	78	72	77	75	72	70
56	THENARASU	35	M	50	GENERAL SURGERY	85	BF	98	96	94	92	90	88	86	86	86	88	84	84	82	80	82	82	94	87	86	84
57	KARUNANIDHI	35	M	50	GENERAL SURGERY	90	BF	76	74	72	68	66	64	64	68	66	68	70	70	70	72	72	70	96	102	92	90
58	CHELLIAYA	46	M	48	GENERAL SURGERY	90	BF	80	82	85	78	76	74	72	70	72	72	72	70	70	74	72	70	94	92	90	90
59	THILAGARESAN	42	M	52	GENERAL SURGERY	75	BF	78	74	72	72	70	70	72	72	70	70		68	66	68	70	70	108	106	100	100
60	ANANDHAN	40	M	50	GENERAL SURGERY	90	BF	88	86	86	84	82	82	80	80	82	82		80	80	82	84	80	104	102	100	96

SL.NO	NAME	MAP 20MINS	MAP 25MINS	MAP 30MINS	MAP 40MINS	MAP 50MINS	MAP 60MINS	MAP 70MINS	MAP 80MINS	MAP 90MINS	MAP 100MINS	MAP 11OMINS	MAP 120MINS	RR 0MINS	RR 5MINS	RR 10MINS	RR 15MINS	RR 20MINS	RR 25MINS	RR 30MINS	RR 40MINS	RR 50MINS	RR 60MINS	RR 70MINS	RR 80MINS	RR 90MINS	RR 100MINS	RR 110MINS	RR 120MINS
1	RAMASAMY	75	73	75	75	78	82	82	86	85	88	90	88	16	16	15	14	15	14	14	14	14	14	15	14	14	14	14	14
2	MANI	72	75	74	76	74	75	79	82	80	85	88	89	16	16	16	15	15	15	14	14	15	15	15	15	16	16	15	16
3	MAHALINGAM	82	80	76	74	74	72	80	82	82	85	90	98	14	14	14	14	12	12	12	12	12	12	12	12	12	13	13	13
4	RAJAMANI	90	83	81	78	76	81	85	82	88	86	91	90	14	14	14	14	14	12	12	12	12	12	12	12	12	12	12	12
5	DHARMARAJ	78	76	74	71	72	74	82	80	84	86	88	88	18	18	18	16	14	14	14	14	13	14	14	14	14	14	14	14
6	RAJASEKAR	86	90	86	84	80	84	84	88	92	90	90	90	16	16	16	16	14	12	12	12	12	12	12	12	12	12	12	12
7	KUMARESAN	88	86	84	82	82	80	85	88	84	85	86	86	14	14	14	14	16	14	14	12	12	12	12	14	12	12	12	12
8	SHANKAR	69	74	74	72	70	70	74	77	78	78	77	78	14	14	12	12	12	14	14	12	12	12	12	14	12	12	12	12
9	SIVAM	82	82	80	79	76	76	74	78	84	86	88	88	16	16	16	16	14	14	14	14	14	14	14	12	12	12	12	12
10	BALASUBRAMANIAN	90	88	85	82	82	84	88	90	88	86	88	88	18	18	18	16	16	14	14	14	14	12	12	12	14	14	14	14
11	SIVASANKAR	88	88	86	86	82	82	80	78	76	74	78	82	18	18	18	16	16	14	14	14	12	12	12	12	12	12	12	12
12	GANAPATHY	92	90	89	86	85	82	82	78	84	86	88	88	16	16	16	16	14	14	14	12	12	12	12	12	12	12	13	12
13	PARVATHI	92	90	85	90	86	84	80	84	86	86	90	92	16	16	16	16	14	14	13	13	12	12	12	12	12	13	12	12
14	AROKIARAJ	86	85	80	82	80	82	82	82	86	88	90	88	16	16	16	16	14	14	12	12	12	12	11	11	11	12	12	12
15	VEDHAMOORTHI	80	81	85	79	80	82	84	84	89	88	90	94	16	16	16	16	14	14	14	12	11	11	11	12	12	12	12	12
16	JESUDAS	84	82	80	85	86	82	86	86	88	90	92	90	14	14	14	14	12	12	12	11	11	12	12	12	12	11	11	12
17	MURUGAIYAN	92	90	90	88	85	82	82	84	84	86	88	92	16	16	16	16	14	14	14	13	13	13	12	12	12	12	11	12
18	RANGANATHAN	71	70	70	72	76	79	79	82	87	85	85	86	14	14	14	14	14	12	12	12	11	11	11	12	12	12	12	12
19	RATHINAM	89	86	84	81	78	80	80	82	84	82	82	84	16	16	16	16	14	14	14	12	12	12	12	12	11	11	12	12
20	BABU	76	76	74	74	72	70	74	74	76	77	80	84	12	12	12	12	12	11	11	10	12	12	12	12	12	12	11	12

SL.NO	NAME	MAP 20MINS	MAP 25MINS	MAP 30MINS	MAP 40MINS	MAP 50MINS	MAP 60MINS	MAP 70MINS	MAP 80MINS	MAP 90MINS	MAP 100MINS	MAP 11OMINS	MAP 120MINS	RR 0MINS	RR 5MINS	RR 10MINS	RR 15MINS	RR 20MINS	RR 25MINS	RR 30MINS	RR 40MINS	RR 50MINS	RR 60MINS	RR 70MINS	RR 80MINS	RR 90MINS	RR 100MINS	RR 110MINS	RR 120MINS
21	KUMARVEL	92	90	88	86	86	84	85	84	86	86	88	88	16	16	16	16	14	14	12	12	12	11	11	12	12	12	12	12
22	VELAUDHAM	77	75	72	72	72	70	74	74	78	80	82	82	14	14	14	14	12	12	11	11	11	12	12	12	12	12	11	12
23	KARNAN	90	88	88	88	86	86	82	84	86	86	88	90	14	14	14	14	14	12	12	12	11	11	11	12	12	12	12	12
24	PAULRAJ	88	84	84	80	78	75	76	80	80	84	86	86	14	14	14	14	14	12	12	12	11	11	11	12	12	12	12	12
25	PETER SELVAM	92	90	90	88	88	86	84	82	84	84	86	88	16	16	16	16	14	14	14	12	12	12	11	11	11	12	12	12
26	SENAPATHY	90	88	83	86	88	88	86	84	86	88	90	90	14	14	14	14	14	13	13	12	12	12	12	11	11	12	12	12
27	GANDHIMADHY	80	78	78	75	75	74	76	76	80	82	84	86	14	14	14	14	14	12	12	12	12	11	11	11	12	12	12	12
28	PETHAN	82	80	80	79	76	74	72	72	76	79	80	82	12	12	12	12	11	11	11	10	11	11	11	12	12	12	12	12
29	VEERAPANDI	74	72	70	70	69	76	78	78	80	80	82	84	14	14	14	14	14	12	12	12	12	11	11	11	11	12	12	12
30	THIYAGARAJAN	76	74	70	70	72	74	74	74	76	76	78	78	12	12	12	12	12	11	11	11	10	10	10	11	11	12	12	12
31	KRISHNAMOORTHY	90	88	83	86	88	88	86	84	86	88	90	90	14	14	14	12	12	12	12	12	12	12	11	11	11	12	12	12
32	RAJANGAM	80	78	78	75	75	74	76	76	80	82	84	86	16	16	16	14	14	14	14	12	12	12	11	11	12	12	14	14
33	MARIMUTHU	82	80	80	79	76	74	72	72	76	79	80	82	16	16	16	14	14	14	13	13	14	14	13	13	14	14	14	14
34	SAIVARAJ	74	72	70	70	69	76	78	78	80	80	82	84	14	14	14	14	13	13	13	13	13	12	12	12	12	12	12	12
35	PANEERSELVAM	76	74	70	70	72	74	74	74	76	76	78	78	14	14	12	12	12	11	11	11	11	11	11	10	12	12	12	12
36	KENNADY	84	82	80	85	86	82	86	86	88	90	92	90	14	14	14	14	14	14	13	13	13	12	12	12	12	12	13	13
37	MAHALINGAM	92	90	90	88	85	82	82	84	84	86	88	92	14	14	14	14	14	14	13	13	12	12	12	12	11	12	12	12
38	KALAVATHY	71	70	70	72	76	79	79	82	87	85	85	86	14	14	14	14	14	14	14	13	13	13	12	12	12	11	12	12
39	SIVASANKAR	89	86	84	81	78	80	80	82	84	82	82	84	16	16	16	16	16	14	14	13	13	13	12	12	12	13	14	14
40	PERUMANANDHAM	76	76	74	74	72	70	74	74	76	77	80	84	14	14	14	14	13	13	12	12	12	11	11	11	13	13	14	14
41	GOVINDARAJ	92	90	88	86	86	84	85	84	86	86	88	88	16	16	16	16	14	14	12	12	12	11	12	12	14	14	12	12

SL.NO	NAME	MAP 20MINS	MAP 25MINS	MAP 30MINS	MAP 40MINS	MAP 50MINS	MAP 60MINS	MAP 70MINS	MAP 80MINS	MAP 90MINS	MAP 100MINS	MAP 11OMINS	MAP 120MINS	RR 0MINS	RR 5MINS	RR 10MINS	RR 15MINS	RR 20MINS	RR 25MINS	RR 30MINS	RR 40MINS	RR 50MINS	RR 60MINS	RR 70MINS	RR 80MINS	RR 90MINS	RR 100MINS	RR 110MINS	RR 120MINS
42	MADHIALAGAN	77	75	72	72	72	70	74	74	78	80	82	82	12	12	12	12	12	12	11	11	11	11	11	11	11	12	12	12
43	PERUMAL	90	88	88	88	86	86	82	84	86	86	88	90	14	14	14	14	14	14	14	12	12	12	12	12	12	12	12	12
44	JOHN	88	84	84	80	78	75	76	80	80	84	86	86	16	16	16	16	14	14	13	12	12	12	12	12	14	14	14	14
45	KATHIRAVAN	92	90	90	88	88	86	84	82	84	84	86	88	14	14	14	14	14	14	12	12	12	12	12	12	12	11	12	12
46	LEELAVATHY	75	73	75	75	78	82	82	86	85	88	90	88	14	14	14	14	14	14	12	12	12	12	12	11	11	11	12	12
47	VELMURUGAN	72	75	74	76	74	75	79	82	80	85	88	89	12	12	12	12	12	12	12	12	11	11	12	12	12	12	12	12
48	PANDIYAN	82	80	76	74	74	72	80	82	82	85	90	98	14	14	14	14	14	14	14	12	12	12	12	12	12	12	12	12
49	MOORTHY	90	83	81	78	76	81	85	82	88	86	91	90	14	14	14	14	14	14	14	14	13	13	13	12	12	12	12	12
50	MURUGESAN	78	76	74	71	72	74	82	80	84	86	88	88	16	16	16	16	16	16	14	14	14	14	12	12	12	14	14	14
51	RAJAMANI	84	82	80	78	77	80	80	84	86	88	90	90	16	16	14	14	14	12	12	12	12	12	11	11	12	12	12	12
52	PETHAIYAN	72	70	70	76	76	76	78	78	76	80	82	84	14	14	14	14	14	14	14	14	14	12	12	12	12	12	13	13
53	THANGARAJ	86	90	86	84	80	84	84	88	92	90	90	90	14	14	14	14	14	14	14	14	13	13	12	12	12	12	12	13
54	VEERAMANI	88	86	84	82	82	80	85	88	84	85	86	86	14	14	14	14	12	12	12	12	12	12	11	11	11	12	12	12
55	KANNAIYAN	69	74	74	72	70	70	74	77	78	78	77	78	16	16	16	16	14	14	14	12	12	12	12	12	13	12	12	12
56	THENARASU	82	82	80	79	76	76	74	78	84	86	88	88	14	14	14	14	14	14	14	14	14	13	13	13	12	12	12	14
57	KARUNANIDHI	90	88	85	82	82	84	88	90	88	86	88	88	16	14	14	14	12	12	12	12	12	12	11	11	10	10	12	12
58	CHELLIAYA	88	88	86	86	82	82	80	78	76	74	78	82	14	14	14	14	14	14	14	14	14	13	13	12	12	12	12	12
59	THILAGARESAN	98	96	94	90	90	88	88	86	86	84	84	82	14	14	14	14	12	12	12	12	12	12	11	11	11	11	12	12
60	ANANDHAN	94	92	90	90	86	84	82	84	80	80	82	82	14	14	14	14	14	14	14	14	14	12	12	12	12	12	11	12

SL.NO	NAME	SPO2 0MINS	SPO2 5MINS	SPO2 10MINS	SPO2 15MINS	SPO2 20MINS	SPO2 25MINS	SPO2 30MINS	SPO2 40MINS	SPO2 50MINS	SPO2 60MINS	SPO2 70MINS	SPO2 80MINS	SPO2 90MINS	SPO2 100MINS	SPO2 110MINS	SPO2 120MINS	SEDATIION SCORE AT 30MINS	SEDATIION SCORE AT 60MINS	SEDATION SCORE AT THE END OF SURGERY	SIDE EFFECTS-NAUSEA/VOMITING	SIDE EFFECTS-PRURITIS	SIDE EFFECTS - RESPIRATORY DEPRESSION	ONSET OF SENSORY BLOCKADE (MINS)
1	RAMASAMY	99	99	99	99	97	97	97	97	97	97	97	97	98	98	98	98	4	4	4	_	_	_	5
2	MANI	99	99	99	98	98	98	98	98	98	97	97	97	98	98	98	98	4	4	5	_	_	_	5
3	MAHALINGAM	99	99	99	98	98	97	97	97	97	97	97	97	98	98	98	98	4	4	4	_	_	_	8
4	RAJAMANI	99	99	98	98	98	98	98	97	97	97	97	97	98	98	99	98	4	4	5	_	_	_	5
5	DHARMARAJ	98	98	98	98	98	98	97	97	97	97	98	98	98	98	98	98	4	4	4	_	_	_	5
6	RAJASEKAR	98	98	98	98	97	97	97	97	97	97	98	98	98	98	98	98	4	4	4	_	_	_	8
7	KUMARESAN	98	98	98	98	98	97	97	97	97	97	98	98	98	98	98	98	4	3	3	_	_	_	7
8	SHANKAR	99	99	99	99	98	98	98	98	98	97	97	97	97	97	97	97	4	4	4	_	_	_	8
9	SIVAM	99	99	99	98	98	98	98	98	98	98	98	98	98	98	98	98	4	4	4	_	_	_	5
10	BALASUBRAMANIAN	98	98	98	98	98	97	97	97	97	97	97	97	97	97	97	98	4	4	4	_	_	_	8
11	SIVASANKAR	98	98	98	98	98	97	97	97	97	97	97	97	98	98	98	98	4	4	4	_	_	_	5
12	GANAPATHY	99	99	99	99	97	97	97	97	97	97	98	98	98	98	98	98	4	4	4	_	Y	_	9
13	PARVATHI	98	98	98	98	98	97	97	97	97	97	97	98	98	98	98	98	4	3	4	_	_	_	5
14	AROKIARAJ	98	98	98	98	97	97	97	97	97	97	97	98	98	98	98	98	4	4	4	_	_	_	5
15	VEDHAMOORTHI	99	99	98	98	98	98	98	97	97	97	97	97	98	98	98	98	4	4	4	_	_	_	5
16	JESUDAS	99	99	99	99	99	98	98	98	97	97	97	97	97	97	97	97	4	4	4	_	_	_	5
17	MURUGAIYAN	99	99	99	99	99	98	98	98	97	97	97	97	98	98	98	98	4	4	4	_	_	_	8
18	RANGANATHAN	98	98	98	98	98	98	98	98	98	97	97	97	98	98	98	98	4	3	4	Y	_	_	9
19	RATHINAM	98	98	98	98	98	98	97	97	97	97	97	97	97	98	98	98	4	4	4	_	_	_	5
20	BABU	98	98	98	98	98	98	98	98	98	98	98	98	98	98	98	98	4	4	4	_	_	_	5

SL.NO	NAME	SPO2 0MINS	SPO2 5MINS	SPO2 10MINS	SPO2 15MINS	SPO2 20MINS	SPO2 25MINS	SPO2 30MINS	SPO2 40MINS	SPO2 50MINS	SPO2 60MINS	SPO2 70MINS	SPO2 80MINS	SPO2 90MINS	SPO2 100MINS	SPO2 110MINS	SPO2 120MINS	SEDATIION SCORE AT 30MINS	SEDATIION SCORE AT 60MINS	SEDATION SCORE AT THE END OF SURGERY	SIDE EFFECTS- NAUSEA/VOMITING	SIDE EFFECTS- PRURITIS	SIDE EFFECTS - RESPIRATORY DEPRESSION	ONSET OF SENSORY BLOCKADE (MINS)
21	KUMARVEL	98	98	98	98	98	98	98	98	97	97	97	97	97	97	98	98	4	3	4	_	_	_	5
22	VELAUDHAM	98	98	98	98	97	97	97	97	97	97	98	98	98	98	98	98	4	4	4	_	_	_	5
23	KARNAN	99	99	99	99	99	98	98	98	98	98	98	98	98	98	98	98	4	4	4	_	_	_	5
24	PAULRAJ	99	99	99	99	99	98	98	97	97	97	97	97	98	98	98	98	4	3	4	_	_	_	5
25	PETER SELVAM	99	99	99	99	98	98	98	98	98	98	98	98	98	98	98	98	4	4	4	_	_	_	8
26	SENAPATHY	99	99	99	99	99	98	98	98	98	98	98	98	98	98	98	98	4	4	4	_	_	_	5
27	GANDHIMADHY	99	99	99	99	99	99	98	98	98	98	98	98	98	98	98	98	4	4	4	_	_	_	5
28	PETHAN	99	99	99	98	98	97	97	97	97	97	98	98	98	98	98	98	4	3	4	_	_	_	5
29	VEERAPANDI	98	98	98	98	98	97	97	97	97	97	97	98	98	98	98	98	4	4	4	_	_	_	5
30	THIYAGARAJAN	98	98	98	98	98	98	97	97	97	97	97	97	98	98	98	98	4	4	4	_	_	_	5
31	KRISHNAMOORTHY	99	99	99	99	98	98	98	98	98	98	98	98	98	98	98	98	5	5	4	_	_	_	5
32	RAJANGAM	99	99	99	99	98	98	98	98	98	98	98	98	98	98	98	98	4	4	5	_	Y	_	5
33	MARIMUTHU	99	99	99	98	98	98	98	98	98	98	98	98	98	98	98	98	4	4	5	_	_	_	5
34	SAIVARAJ	99	99	99	99	98	98	98	97	97	97	98	98	98	98	98	98	5	5	5	_	_	_	5
35	PANEERSELVAM	98	98	98	98	98	97	97	97	97	97	97	97	98	98	98	98	4	4	4	_	_	_	8
36	KENNADY	98	98	98	98	98	98	97	97	97	97	97	97	97	98	98	98	4	4	4	_	_	_	5
37	MAHALINGAM	98	98	98	98	98	97	97	97	97	97	97	97	98	98	98	98	5	4	4	_	_	_	5
38	KALAVATHY	98	98	98	98	98	98	98	98	98	97	97	97	97	97	97	98	4	4	4	_	Y	_	6
39	SIVASANKAR	98	98	98	98	98	98	97	97	97	97	97	97	97	97	98	98	4	4	4	_	_	_	10
40	PERUMANANDHAM	98	98	98	98	98	98	97	97	97	97	97	98	98	98	98	98	5	4	4	Y	_	_	5
41	GOVINDARAJ	99	99	99	99	99	98	98	98	98	98	98	98	98	98	98	98	4	4	4	_	_	_	5

SL.NO	NAME	SPO2 0MINS	SPO2 5MINS	SPO2 10MINS	SPO2 15MINS	SPO2 20MINS	SPO2 25MINS	SPO2 30MINS	SPO2 40MINS	SPO2 50MINS	SPO2 60MINS	SPO2 70MINS	SPO2 80MINS	SPO2 90MINS	SPO2 100MINS	SPO2 110MINS	SPO2 120MINS	SEDATIION SCORE AT 30MINS	SEDATIION SCORE AT 60MINS	SEDATION SCORE AT THE END OF SURGERY	SIDE EFFECTS- NAUSEA/VOMITING	SIDE EFFECTS- PRURITIS	SIDE EFFECTS - RESPIRATORY DEPRESSION	ONSET OF SENSORY BLOCKADE (MINS)
42	MADHIALAGAN	99	99	99	99	99	99	98	98	98	98	98	98	98	98	98	98	5	5	5	_	Y	_	5
43	PERUMAL	99	99	99	99	99	99	99	99	99	99	98	98	98	98	98	98	4	4	4	_	_	_	6
44	JOHN	99	99	99	99	99	98	98	98	98	98	98	98	98	98	98	98	4	4	4	_	_	_	10
45	KATHIRAVAN	99	99	99	99	98	98	98	98	98	98	98	98	98	98	98	98	5	4	4	_	_	_	5
46	LEELAVATHY	99	99	99	98	98	98	98	98	98	98	98	98	98	98	98	98	5	4	4	_	_	_	5
47	VELMURUGAN	99	99	99	99	99	99	98	98	98	98	98	98	98	98	98	98	4	4	4	_	Y	_	10
48	PANDIYAN	99	99	99	99	99	99	98	98	98	98	97	97	97	97	98	98	4	4	4	_	_	_	5
49	MOORTHY	99	99	99	99	98	98	97	97	97	97	97	97	97	98	98	98	5	5	5	Y	_	_	5
50	MURUGESAN	99	99	99	99	99	98	98	98	98	98	98	98	98	98	98	98	4	4	4	_	_	_	5
51	RAJAMANI	99	99	99	98	98	98	98	97	97	97	97	97	98	98	98	98	5	4	4	_	_	_	5
52	PETHAIYAN	99	99	99	98	98	98	98	97	97	97	97	97	97	98	98	98	4	4	4	Y	_	_	5
53	THANGARAJ	99	99	99	99	99	99	99	98	98	98	98	98	98	98	98	98	5	4	4	_	_	_	5
54	VEERAMANI	99	99	99	99	99	99	99	99	98	98	98	98	98	98	98	98	4	4	4	_	_	_	8
55	KANNAIYAN	99	99	99	99	99	99	98	98	98	98	98	98	98	98	98	98	4	4	4	_	Y	_	8
56	THENARASU	99	99	99	99	99	98	98	98	97	97	97	97	97	98	98	98	5	4	4	_	_	_	5
57	KARUNANIDHI	99	99	99	98	98	98	98	98	98	98	98	98	98	98	98	98	5	5	5	_	_	_	5
58	CHELLIAYA	99	99	99	98	98	98	98	98	98	97	97	97	97	97	98	98	5	4	4	_	_	_	5
59	THILAGARESAN	99	99	99	99	99	99	99	99	99	98	98	98	98	98	98	98	4	4	4	_	Y	_	5
60	ANANDHAN	99	99	99	99	98	98	98	98	98	98	98	98	98	98	98	98	4	4	4	_	_	_	5

SL.NO	NAME	COMPLETION OF SENSORY BLOCKADE (MINS)	ONSET OF MOTOR BLOCKADE(MINS)	LEVEL OF ANALGESIA	QUALITY OF ANALGESIA	DURATIO OF ANALGESIA (HRS)
1	RAMASAMY	10	4	T6	GOOD	7
2	MANI	10	4	T6	GOOD	7
3	MAHALINGAM	12	4.5	T6	GOOD	7.5
4	RAJAMANI	10	4.5	T6	GOOD	5.5
5	DHARMARAJ	9	4.5	T8	GOOD	7
6	RAJASEKAR	12	4.5	T6	GOOD	5.5
7	KUMARESAN	8	4.5	T6	GOOD	6.5
8	SHANKAR	10	4.5	T6	GOOD	6.5
9	SIVAM	10	4	T6	FAIR	7.5
10	BALASUBRAMANIAN	12	4	T6	GOOD	7.5
11	SIVASANKAR	10	4	T8	GOOD	7.5
12	GANAPATHY	8	4	T6	GOOD	7.5
13	PARVATHI	10	4.5	T6	GOOD	5
14	AROKIARAJ	10	4.5	T6	GOOD	7
15	VEDHAMOORTHI	8	4.5	T6	GOOD	7.5
16	JESUDAS	10	4.5	T8	GOOD	7
17	MURUGAIYAN	9	4.5	T6	GOOD	7.5
18	RANGANATHAN	12	4.5	T6	GOOD	7
19	RATHINAM	10	5	T6	GOOD	7
20	BABU	12	5	T6	GOOD	7

SL.NO	NAME	COMPLETION OF SENSORY BLOCKADE (MINS)	ONSET OF MOTOR BLOCKADE(MINS)	LEVEL OF ANALGESIA	QUALITY OF ANALGESIA	DURATIO OF ANALGESIA (HRS)
21	KUMARVEL	9	4	T8	GOOD	7
22	VELAUDHAM	10	4.5	T6	GOOD	7
23	KARNAN	10	4.5	T6	GOOD	7
24	PAULRAJ	12	5	T6	GOOD	6.5
25	PETER SELVAM	10	4.5	T8	GOOD	6.5
26	SENAPATHY	8	4.5	T8	GOOD	10
27	GANDHIMADHY	12	4.5	T6	GOOD	10
28	PETHAN	10	5	T6	GOOD	7.5
29	VEERAPANDI	10	4.5	T6	GOOD	7.5
30	THIYAGARAJAN	10	4.5	T6	GOOD	7.5
31	KRISHNAMOORTHY	10	5.5	T6	GO	5
32	RAJANGAM	10	5.5	T6	GOOD	5
33	MARIMUTHU	12	5.5	T6	GOOD	3.5
34	SAIVARAJ	10	6	T6	GOOD	5
35	PANEERSELVAM	10	6	T6	GOOD	5
36	KENNADY	10	6	T6	GOOD	5
37	MAHALINGAM	12	6	T6	FAIR	5.5
38	KALAVATHY	10	5.5	T6	GOOD	5
39	SIVASANKAR	12	5.5	T6	GOOD	5.5
40	PERUMANANDHAM	10	5	T6	GOOD	8
41	GOVINDARAJ	10	5	T8	GOOD	8

SL.NO	NAME	COMPLETION OF SENSORY BLOCKADE (MINS)	ONSET OF MOTOR BLOCKADE(MINS)	LEVEL OF ANALGESIA	QUALITY OF ANALGESIA	DURATIO OF ANALGESIA (HRS)
42	MADHIALAGAN	12	5	T6	GOOD	5
43	PERUMAL	10	5	T6	GOOD	5.5
44	JOHN	10	5.5	T6	GOOD	5.5
45	KATHIRAVAN	10	5.5	T6	GOOD	5
46	LEELAVATHY	10	5.5	T6	FAIR	5
47	VELMURUGAN	10	5.5	T6	GOOD	5
48	PANDIYAN	10	5.5	T6	GOOD	8
49	MOORTHY	12	5.5	T6	GOOD	5
50	MURUGESAN	10	5.5	T8	GOOD	5
51	RAJAMANI	10	5.5	T6	GOOD	5
52	PETHAIYAN	12	5.5	T6	GOOD	5
53	THANGARAJ	12	6	T6	GOOD	5.5
54	VEERAMANI	10	5.5	T6	GOOD	5
55	KANNAIYAN	10	5.5	T6	FAIR	5
56	THENARASU	10	5.5	T6	GOOD	4.5
57	KARUNANIDHI	10	5.5	T6	GOOD	3.5
58	CHELLIAYA	10	5	T6	GOOD	3.5
59	THILAGARESAN	12	5	T6	GOOD	5
60	ANANDHAN	10	5.5	T6	GOOD	5.5